Canadian Medical Association

The Canadian Medical Association Journal

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Published by

THE CANADIAN MEDICAL ASSOCIATION

1936

JUL 8 - 1936

VOL. 35 TORONTO, JULY, 1936 No. 1

MEDICAL LIBRARY

The Canadian Medical Association Journal

THE OFFICIAL ORGAN OF
THE CANADIAN MEDICAL ASSOCIATION
AND ITS AFFILIATED PROVINCIAL MEDICAL ASSOCIATIONS

PUBLISHED MONTHLY BY
THE CANADIAN MEDICAL ASSOCIATION
3640 UNIVERSITY STREET, MONTREAL

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The Canadian
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Vol. 35 TORONTO, JULY, 1936 No. 1

CONGENITAL LUNG CYSTS*

BY J. D. ADAMSON, M.D., M.R.C.P.(EDIN.), F.R.C.P.(C.),

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IN 1687 Bartholin described a cystic lung. Since then pathologists have from time to time reported similar cases. Almost all of the illustrious German pathologists of the past century have contributed to the subject. In 1925 Koontz[1] collected the accounts of 108 cases of what he regarded as congenital cystic lungs. All of these were discovered at autopsy.

The clinical recognition of this condition has become possible only since radiography and bronchoscopy have come into common use. It is indeed only in the past ten years that antemortem diagnosis has been made. The earliest clinical case was reported by Miller[2] in 1926. Since that time many articles have appeared and about 50 cases have been diagnosed on clinical and x-ray findings. Many of these have been confirmed by autopsy. The condition can therefore be said to have ceased to be a mere pathological curiosity and to have become a clinical entity of importance. It must be kept constantly in mind when making a differential diagnosis in chronic pulmonary disease of any sort. Interest is enhanced by the fact that almost any chronic lung condition may be simulated. Cases have been mistaken for, and reported as, chronic pneumothorax, chronic cavitating tuberculosis, chronic pleurisy with effusion, diaphragmatic hernia, chronic empyema, chronic abscess, neoplasm, etc. Four new cases will be reported in this communication, and then classification, clinical characteristics, and pathogenesis will be briefly discussed.

* Read at the Annual Meeting of the Canadian Tuberculosis Association, June, 1935.

CLINICAL CASES

CASE 1.—SIMULATING CHRONIC BILATERAL PNEUMOTHORAX

This case is of particular interest because, since the man is a pensioner, we have authentic written medical records that go back to 1915 and x-ray films dating back to 1925.

There is nothing significant in his family history, and he recollects no disabling illness until 1915. He then had cough and expectoration for two months, but seems not to have been seriously incapacitated. Since then he has been subject to occasional attacks of "bronchitis". He joined the army in 1915, and performed the ordinary duties of a private with no particular complaint. On demobilization in 1918 he returned to work as a motor mechanic. He continued this without undue distress, though he frequently had some cough and expectoration.

In January, 1925, the cough and expectoration became worse and he was suddenly seized with a sharp pain in the right chest, and dyspnœa became marked. He was then hospitalized for five months by Dr. B. H. Olson with a diagnosis of spontaneous pneumothorax. The film (Fig. 1), taken at this time shows what appears to be a right pneumothorax and a partial left pneumothorax. It was diagnosed as such and presumed to be tuberculous, though frequent sputum examination showed no tubercle bacilli. The physical signs were those of pneumothorax. He recovered from the urgent dyspnœa, and from 1925 to 1930 carried on as a motor mechanic. During this time he had slight cough and expectoration, and definite dyspnœa on exertion.

In 1928 he developed a pleural effusion which apparently gave him very little trouble and gradually disappeared (Fig. 2). Since that time his condition has not changed much, though dyspnœa has increased, and he has done no steady work since 1933. He has not been laid up and has surprisingly little dyspnœa on ordinary activity. X-rays taken from time to time showed the appearance of double pneumothorax which alternated to some extent from one side to the other.

In 1934 (Fig. 3) he was hospitalized for a time, largely for purposes of investigation. Physical examination at that time gave evidence of very voluminous hyper-resonant and relatively silent lungs. An effort was made to draw off air from the left side. The pressure was found to oscillate from plus 2 to minus 2, and was not altered by removing air. After this simple procedure he felt ill for the first time during the course of his disease. This was due to a small quantity of fluid having been developed as a result of the puncture.

On this account we refrained from any further special investigation, which would certainly not be without danger in one with so little functioning lung. Since that time his condition has not changed. Dyspnœa on effort is his only complaint.

The diagnosis, I think, is clear for the following reasons:—

1. Double pneumothorax of this degree and duration could only exist in the presence of a double broncho-pleural fistula. It is highly improbable that this could persist for so long without having produced infection of one or other pleural sac before now. There has never been any evidence of this having occurred, and he has at no time had fever or any other evidence of infection.

2. The effusion which he developed was encysted in the pleural space and did not have a horizontal level—as in a pneumothorax. The device of putting air into the pleural sac in cases suspected of cystic disease has been resorted to by others in order to prove that the apparent pneumothorax is actually inside the lung itself. Some cases have been proven by this method. In this case the fortuitous development of fluid has confirmed the diagnosis for us.

3. This case is identical in x-ray findings with several in the literature that have been proved to be cysts by post-mortem and other means. A similar case is reported by Crosswell and King.[9] Wood[10] also describes a large single cyst on the right which herniated into the left side of the chest. Four of Koontz's cases also appeared to be single cysts which encroached on both lungs. It is possible, therefore, that the case here reported is a single cyst with a mediastinal hernia.

4. The fourth reason for believing this to be cystic is that tubercle bacilli have never been found, and no other evidence of tuberculosis exists.

5. The cysts are of a shape entirely different from the contour of pneumothorax, and at no time is there seen any shadow of collapsed lung. Very large cysts of this sort have heretofore been described almost exclusively in young children. Many of them are reported as "chronic or recurring double pneumothorax."

CASE 2.—SIMULATING CHRONIC PULMONARY TUBERCULOSIS WITH CAVITATION

This case is also that of a soldier, with an authentic documentary history going back twenty years. His age is now 41. There is a history of recurring cough and expectoration dating back to the age of 3. This rendered him unfit to indulge in the ordinary activities of boyhood.

He enlisted in 1914 at the age of 21, and was in France for five months. He was then invalided out with a diagnosis of pulmonary tuberculosis. He was sent to Pinewood Sanatorium. His physical signs were as follows: "Dullness throughout right upper half with broncho-vesicular breathing, and coarse crepitations which were increased on cough." These same signs have been present unchanged ever since. After six months' observation the physician in charge said: "It seems probable that this case has been wrongly diagnosed, and is really one of bronchiectasis". This conclusion was arrived at because, though there was fairly profuse expectoration, no tubercle bacilli were found, and because, though there were quite gross and widespread signs, the general symptoms of tuberculosis were not present. In the light of subsequent developments this was a very astute conclusion. Perhaps if that physician had had x-rays to help him, he would have made the same mistakes as many of us made later.

Since that time, twenty years ago, the man has been examined by at least a dozen reputable chest specialists and physicians, and has been x-rayed by almost as many radiologists. Until recent years the diagnosis has always been gross pulmonary tuberculosis. Doubts have been expressed on several occasions. No other diagnosis, however, could be made in the presence of typical signs of fibroid tuberculosis. Until recently there was no other recognized cause for such a clinical picture. (Figs. 4 and 5).

In the light of recent reports on congenital cystic disease it seems certain that this is such a case. The chief reasons for making this diagnosis are:—

(1) The trouble definitely dates from early boyhood. (2) Symptoms and physical signs have not changed in the slightest over a period of twenty years of observation. (3) Tubercle bacilli have at no time been found—in spite of fairly profuse expectoration. (4) In spite of gross signs he has never had any constitutional evidences of tuberculosis. He has, in fact, never been laid up, though often a little below par. (5) In 1924 he had throughout the right side a loud pleural rub. This was reported by Dr. W. L. Mann on several occasions, and he seemed then at a loss to reconcile it with the fact that the man had no other subjective or objective evidence of a pleurisy. This observation of a loud pleural rub over a cystic lung has been made by at least two other observers (Oechsli and Miles: Stewart *et al.*). It has been suggested that its presence is of some diagnostic value. Stewart *et al.*[6] report such a case in which post-mortem showed no evidence of pleurisy. (6) The x-ray plates have not changed in ten years. Also they have quite a different appearance from those of the usual tuberculous case: the honeycombing is far too symmetrical and no definite parenchymal shadows are seen. (7) Gross tuberculous infection of such long duration would unquestionably have given rise to some retraction. There is no evidence of this on examination or on the

plates. In fact there is definite bulging over the right upper chest. (8) This case could be quite properly regarded as one of congenital bronchiectasis; indeed it is likely that defective drainage makes all cysts clinically identical with bronchiectasis.

CASE 3.—SIMULATING A LARGE PLEURAL EFFUSION

A French Canadian woman of 37 was admitted to St. Boniface Sanatorium on February 1, 1932. She gave a history of having had influenza in 1919, since when she has had slight cough and expectoration which has never laid her up. In 1920 she had what was diagnosed as pleurisy with effusion, and was confined to bed for two weeks. No aspiration was done and she made a good recovery. From 1920 to 1928 she did ordinary house work and bore a child about once a year. She was frail and thin, but had no particular complaint except occasional cough and expectoration. In 1928 she began to become weak and dyspnœic. This gradually increased. In 1930 she was in St. Boniface Hospital for some weeks. X-rays (Fig. 6) showed opacity over the whole of the right side, with much pushing over of the mediastinum, so that the left lung was compressed to less than one-half of its normal size. Several attempts at aspiration produced only small quantities of thick gelatinous material. She left the hospital slightly improved, but the x-ray picture showed no change. Thereafter her cough improved, but dyspnœa became worse.

In February, 1932, she was admitted to St. Boniface Sanatorium complaining of slight cough and about one dram of expectoration, slight pain between the shoulders and dyspnœa, though only on fast walking. She had physical signs of gross pleural effusion with much mediastinal distortion. The plate was identical with that taken 20 months before (Fig. 6). She remained in the sanatorium 8 months. During that time 3,500 c.c. of thick gelatinous material were withdrawn in 10 separate aspirations and replaced with air. The report on the fluid was—"A gelatinous coagulum, turbid and dark yellow, with granular debris and numerous fatty acid and cholesterol crystals: cells are scanty, consisting of an occasional polymorphonuclear and unidentified larger cells". On each aspiration large negative pressures were created. Enough air was run in on each occasion to restore it to atmospheric pressure. After the gelatinous material was completely displaced by air (Fig. 7), it was seen that the thoracic cavity was traversed by dense trabeculæ. Close to the mediastinum there were several grape-like opacities. There was no evidence of lung tissue compressed or otherwise. What appeared to be herniation of the mediastinum persisted even after the material was removed. The patient was allowed to go home, and she was then symptom-free. She was examined again on August 1, 1935, when her general health was good and she denied symptoms. X-ray, however, showed the same condition as is shown in the first plate. This appeared to be a large multilocular cyst which is not in communication with the bronchial tree, and in which bronchial secretions accumulate.

The only similar cases that I have found in the literature were in infants (Wolman,[3] Swansen *et al.*).

CASE 4.—SIMULATING DIAPHRAGMATIC HERNIA

A woman, 70 years of age, was admitted to the Winnipeg General Hospital in February, 1935, with chronic renal disease, from which she died. During the course of cardiac investigation an x-ray was taken of her chest. It showed what was first thought to be a diaphragmatic hernia, with the stomach in the left thoracic cavity (Fig. 8). However, a plate taken with barium in the œsophagus and stomach showed this not to be the case, and an ante-mortem diagnosis of pulmonary cyst was made by Dr. J. C. McMillan. The patient died soon after, and an autopsy showed a very large cyst occupying the lower half of the left lung. It contained no fluid, but no definite communication with the bronchi could be demonstrated. Its inner surface was unpigmented, smooth and trabeculated, as is usual with congenital cysts.

Her history disclosed no evidence of any respiratory disease or disability at any time, and there was no evidence that the cyst had contributed to her death.

CLASSIFICATION

Anspach and Wolman[8] have classified cysts, first, as to whether or not they contain air or fluid, and have subdivided each of these groups again into those that communicate with the bronchial tree and those that do not. This subdivision seems unjustified. It seems reasonable to assume that if the cyst is filled with secretion it does not communicate freely with the bronchial tree, and if it contains air it does communicate. This is the assumption we always work on when dealing with any other cavitation of the lung (*e.g.*, tuberculous cavities, abscesses and bronchiectasis). It is questionable whether any lung cavity could contain air or gas except when in communication with the air passages. In any event the presence or absence of communication is almost impossible to be sure of, even on autopsy. Certainly the only reasonable clinical criterion of communication must be the presence of air.

Having divided cysts into those that contain air and those that contain fluid, the only further subdivision must be as to the size, number and position of the cysts. In order to learn something in connection with these details a study of 100 cases has been made, 51 from Koontz's article and 49 that have been reported since (including the 4 in this article). A partial summary of the results of this analysis appears in Table I.

TABLE I

	From Koontz	*Since*	*Total*
A. Single cysts	22%	50%	36%
B. Multiple	8% right	10% right	9%
1. One lobe	8% left	0 left	4%
2. One lung	6% right	6% right	6%
	8% left	8% left	8%
3. Both lungs	48%	26%	37%

In this analysis only 51 of Koontz's 108 cases were used. The pure aplasias, anatomical anomalies, and diverticulæ were not included, because they cannot be considered as coming into the same group clinically, though they are no

doubt closely related developmentally and pathologically. Only 49 cases that have been reported in the past 10 years were included, since some did not give all the necessary details. Of these about one-half had the diagnosis confirmed by autopsy, and the other half presented convincing clinical and x-ray evidence.

It is found that the size of lung cysts varies from those that are microscopic to those that are so large as to cause death by encroachment on lung tissue. The number also varies greatly. About one-third of all the cases are solitary. These are divided practically equally on the two sides, but show a definite tendency to originate from the upper lobes. The distribution of multiple cysts seems haphazard. Thirteen per cent are limited to one lobe. The right side shows a preponderance in cases reported in recent years, and the right upper lobe is a common site. Fourteen per cent involve the whole of one lung, both sides being equally vulnerable. Both lungs are involved in 37 per cent of cases. Roughly, it may be said in summary that about one-third of the cases are solitary cysts and two-thirds are multiple. Of those that are multiple one-half are found on both sides and one-half are limited to one lobe or one side. This may be represented schematically as follows:

Cysts { Solitary 1/3
{ Multiple 2/3 { Bilateral 1/3
{ Unilateral 1/3 { One lobe 1/6
{ One lung 1/6

CLINICAL FEATURES

The 49 recent cases referred to above have been examined to discover clinical characteristics.

Sex incidence.—There were almost twice as many males as females. This differs from Koontz's figures which show the sex incidence to be equal.

Age incidence.—Cases are discovered throughout the whole span of life. If still-born children and those who died during the first year are left out, the average age is 28. The oldest case (70 years) is reported above (Case 4). The percentage of cases in each decade is shown below:

Infants and still-born, 20 per cent; first decade, 17 per cent; second decade, 10 per cent; third decade, 12 per cent; fourth decade, 11 per cent; fifth decade, 22 per cent; sixth decade, 6 per cent; seventh decade, 2 per cent.

This widespread age incidence with a preponderance in the new-born agrees with what Koontz has found.

Clinical groups.—The cases fall into several groups according to their resemblance to other well known clinical conditions. Indeed most of the cases were at first mistaken for the conditions which they chanced to simulate. The following list gives the percentage of cases which strongly resembled the clinical entities indicated:

	Percentage
Spontaneous pneumothorax	40
Empyema	16
Pulmonary tuberculosis	14
Bronchiectasis	10
"Blue babies"	10
Emphysema	4
Hydrothorax	4
Eventration	2

Symptoms.—Many of the patients lead a long and useful life without symptoms (*e.g.*, patient No. 4); others are still-born or die of cyanosis and dyspnœa immediately after birth. Between these extremes one finds symptoms of every grade of severity. Dyspnœa is commonest and was present in 60 per cent of cases. Cough and expectoration was found in 44 per cent, cyanosis in 22 per cent, and hæmoptysis in 4 per cent. Sixteen per cent had classical symptoms of gross pulmonary sepsis ("congenital bronchiectasis").

PATHOGENESIS

The pathogenesis of pulmonary cysts is obscure. It is generally agreed that they are fundamentally due to congenital defects. This is attested by the following facts. (1) They are quite common in the new-born and in premature children. (2) They are not infrequently associated with other congenital anomalies. (3) The cells of these cysts are not pigmented, which would indicate that they have arisen from tissues that have not functioned as normal lung tissue. (4) They are common in anomalous and supernumerary lobes or lungs.

When considering pathogenesis we must at the outset divide cysts into two groups: (1) those which are not connected with the bronchial tree; and, (2) those which are obviously a part of it.

The cysts of the first group are filled with mucoid secretion, and are seen on x-ray plates as globular opacities if they are small, and diffuse opacities if they are large. The most plausible explanation for their origin seems to be that which assumes that during early embryonic life one or more buds of the developing trachea are pinched off and develop autonomously. Being unconnected with the bronchial tree,

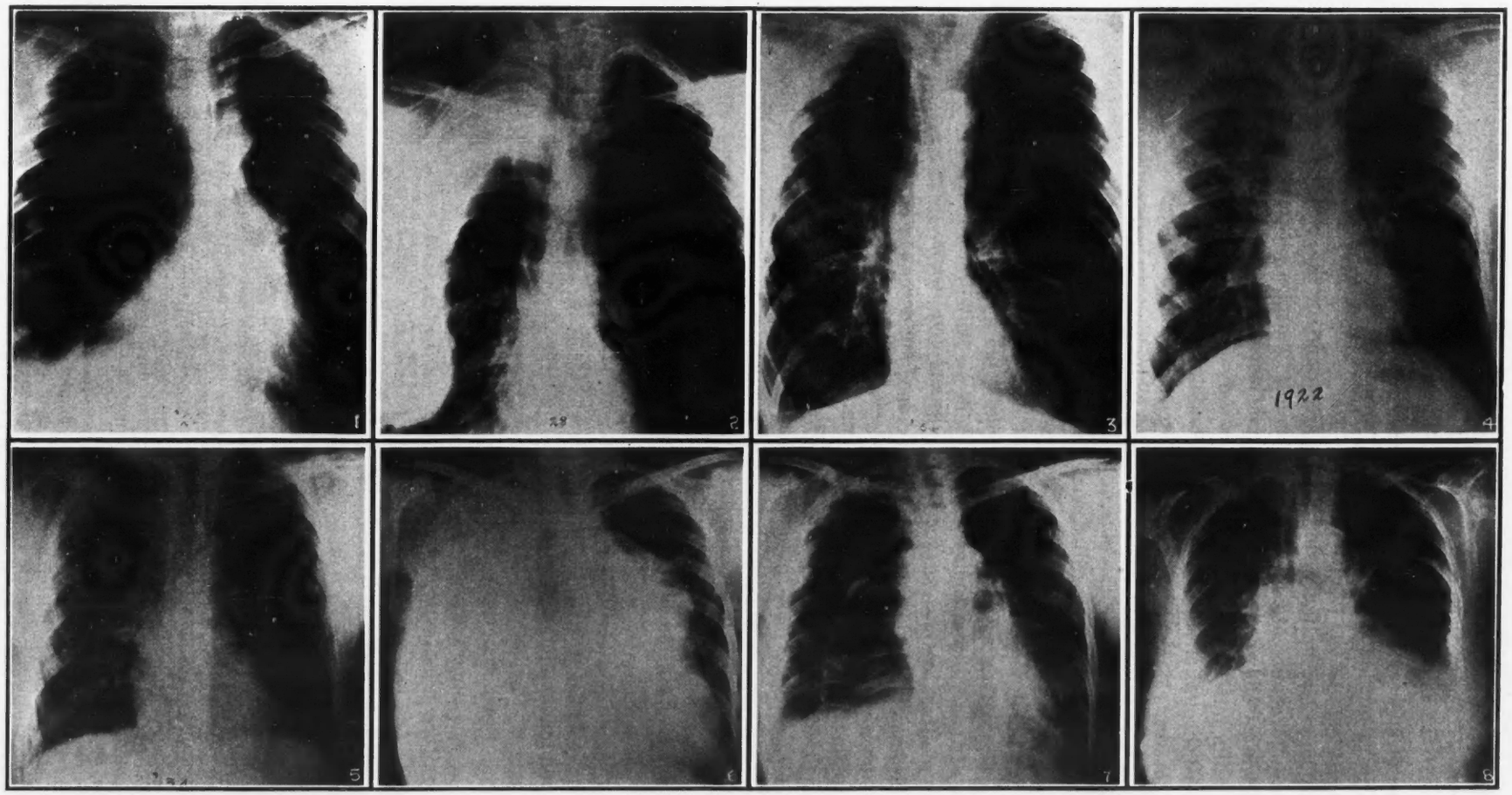

Fig. 1 (Case 1).—Taken in 1925; showing partial obliteration of both lung fields by air-containing cysts. **Fig. 2** (Case 1).—Taken in 1928; showing effusion in pleural sac and cysts in lung. **Fig. 3** (Case 1).—Taken in 1934; cystic condition now more marked in the right lung. **Fig. 4** (Case 2).—Taken in 1922; showing cystic honeycombing throughout the right upper lobe. **Fig. 5** (Case 2).—Taken in 1934; no change has taken place in twelve years. **Fig 6** (Case 3).—Taken in 1930; showing large cyst or cysts full of secretion occupying three-quarters of the thoracic space. **Fig. 7** (Case 3).—Taken in 1932; showing large multilocular cyst after withdrawal of fluid contents. **Fig. 8** (Case 4).—Showing large cyst in left lower lobe, simulating diaphragmatic hernia.

the secretions accumulate and form cysts. They often attain such a size as to compress the whole of one or both lungs. Case 3 comes into this category.

A second source of such cysts may be atresia of a bronchus in intra-uterine life. This could conceivably isolate a part of the lung from the air spaces. It is, however, unlikely that this could occur to any large extent, since it has been fairly convincingly shown that the alveoli are not isolated units but communicate freely with one another throughout each lobe by vents, pores or fenestræ (Macklin). Anything less than a lobar atresia cannot in itself cause isolation because of these vents in the alveoli.

Most cysts that one encounters are filled with air, and a great many of these are obviously in communication with the bronchial tree. Speculation as to their origin is more complex than in the first group. It may be that some of them arise in the same manner as those of the first group, *i.e.*, by the accidental detachment and isolation of small buds of the developing lung. Later they break through into the bronchial tree, discharge their mucoid contents, and become filled with air. A good case for such a series of events is made out in some individual cases (Wolman[3]). Also individual cases have been described in which some of the cysts are full of air and some contain mucoid secretions of various sorts or pus.

A more likely explanation for these air-containing cysts is that they result from a failure in the development of the alveolar part of the lung. This growth does not take place normally until the third trimester of intra-uterine life, and there is a good deal of evidence to show that its failure is not uncommon. In that event the atria are left as open passages ending blindly. Such a situation may be the beginning of congenital bronchiectasis, as Sauerbruch suggests. These blind saccules may also account for many of the air-filled cysts. Since their collateral air circulation is cut out on account of absence of alveolar air vents, various degrees of inflation could take place.

To account for the very large air cysts which are found in the very young and which commonly cause death by displacement of the mediastinum, one must assume the existence of a valve opening into one of these atria. Some histological reports of these cysts suggest the existence of a cellular redundance at the mouth of bronchioles which could cause such a valve action.

SUMMARY

1. Cystic disease of the lung is not uncommon.

2. It usually simulates other well known clinical conditions.

3. Four new cases are reported.

4. Classification, clinical characteristics, and pathogenesis are discussed after a study of about 100 cases in the literature.

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PATHOLOGICAL INTERPRETATIONS OF ROENTGENOLOGICAL SHADOWS IN PNEUMOCONIOSIS.—H. C. Sweany has attempted a correlation of the ante-mortem and post-mortem roentgenograms with the pathological changes in various unclassified types of pneumoconiosis. Tuberculosis with silicosis renders a roentgenographic examination difficult or impossible in the majority of patients affected with silicosis, because of atypical characteristics or the location of the lesions. The localization resulting from tuberculosis may be predominantly that of a hæmatogenous tuberculosis, situated bilaterally in the upper parts of both lungs, of a bronchogenic spread bilaterally, or of a bronchogenic spread unilaterally, and even along a single bronchial ramus. The involvement of the lymph nodes is quite characteristic, forming what have been termed "eggshell" calcifications. Pneumoconiosis, resulting from a lymphatic congestion due to coal, iron and the like, differs from silicosis and silicotuberculosis sufficiently that an involvement by such inert dust may be anticipated many times on the ante-mortem roentgenograms. When the inert dusts are combined in some yet undetermined proportions with a silicosis or a silicotuberculosis, it seems to result in a complex that possesses a variety of bizarre formations, some of which simulate single or multiple neoplastic masses on the roentgenograms.—*J. Am. M. Ass.*, 1936, **106**: 1959.

CARCINOMA OF THE LARYNX, A PLEA FOR MORE CONSERVATIVE SURGERY IN CERTAIN CASES WHERE LARYNECTOMY MIGHT BE CONSIDERED NECESSARY*

BY NORMAN PATTERSON, M.B., CH.B., F.R.C.S. (ENG.),

London, Eng.

WE should remember in treating neoplasms of the larynx that it is not a structure entirely concerned in respiration and vocalization. A portion only of the organ is directly responsible for voice production, and there are regions intimately connected with the act of swallowing, namely, the epiglottis, the ary-epiglottic folds, and the mucous membrane covering the posterior part of the cricoid ring and its muscles. The pyriform fossa is also in relationship externally to a small portion of the thyroid wing and is a recess of the pharynx rather than of the larynx. Growths confined to these regions can often be approached via the pharynx, and the operation entails a minimum of interference with the respiratory and vocal functions. It must be admitted that most growths of the pyriform fossa and all but early tumours of the retro-cricoid pharynx require a laryngo-pharyngectomy for their complete eradication. As I have already stated, however, in Toronto, it is questionable whether such procedures are justifiable. Here I may again say that the term "laryngectomy" appears to me to be somewhat inaccurate, as it is impossible to remove the whole larynx without taking away a small portion, at any rate, of the pharynx. In localized growths affecting the uppermost regions of the larynx and in small tumours involving the retrocricoid pharynx, lateral pharyngotomy is the ideal method of approach, although a very circumscribed growth on the epiglottis or ary-epiglottic fold may sometimes be excised or destroyed by diathermy through the mouth.

A limited cancer of the vocal cord situated in the usual site about the middle or just anterior to the middle of the cord is an ideal case for thyro-fissure, (a better term than laryngo-fissure which would indicate splitting of the cricoid as well as the thyroid).

It is rare to meet with a growth developing from the false cord or ventricle, but such a tumour if superficial might be treated by the same method. If the cord is movable and if at operation there is found to be little tendency to infiltration it would seem to me advisable not to remove the overlying cartilage, as it acts as a barrier should recurrence take place, causing delay in involvement of the soft tissues of the neck and rendering complete extirpation easier to carry out. If the growth crosses the middle line and has spread to the opposite cord or subglottic region, or if it invades the region of the anterior commisure, I do not consider the case a suitable one for thyrotomy. So long, however, as it falls short of the arytenoid region a laryngo-pharyngectomy is generally unnecessary. When the growth has extended to, or is confined to, the subglottic region, but still occupies a position anterior to the transverse axis of the larynx, a partial or anterior laryngectomy, rather than a complete extirpation, will often be sufficient. I shall shortly describe the methods I have employed in such cases. Operations on the lines I shall mention are favoured by certain British and French laryngologists. The operation was developed, if not actually originated, in England by my predecessor at the London Hospital, Lambert Lack, and with sundry modifications I have found it admirably adapted, broadly speaking, for cases of cancer of the larynx situated anteriorly—cases which are, however, unsuitable for thyrotomy because of bilateral involvement or subglottic extension. Unfortunately, cases suitable for this type of operation are not very commonly met with. A very similar procedure is described by Hantant, Portmann and others, but to my mind the operation is too formal and not elastic enough to cope with the varying conditions which may be present in the larynx; it is indeed a hemilaryngectomy. The operation should be so planned as to adequately remove

* Read at the combined Annual Meetings of the Canadian and American Medical Associations at Atlantic City, on June 12, 1935, Section on Laryngology, Otology and Rhinology.

the disease with a sufficient margin of healthy tissue and no more; not a particular portion of the larynx.

It will now be my endeavour to describe the operation in some detail. The preliminary preparations are exactly the same as for thyrofissure. Attention should be paid beforehand to the elimination, as far as possible, of buccal, pharyngeal and nasal sepsis. It will usually be necessary for the patient to undergo preliminary dental treatment unless he is already edentulous, and it may be deemed advisable in some cases to remove all the teeth. The patient should enter the hospital or nursing home two or three days before the operation is to take place. If there is doubt that the disease can be removed by the operation I will shortly describe consent should be obtained for a complete extirpation. The best anæsthetic is chloroform, or gas and oxygen, the latter having some distinct advantages. No preliminary medication in the form of nembutal, avertin or morphia is allowable—the latter is highly dangerous. Anæsthesia should never be deep. Some surgeons will prefer to perform the operation under local anæsthesia, either by the infiltration method or by blocking the nerves supplying the region. The patient should lie with his head well extended and not tilted to either side, or he may be placed in the Trendelenberg position. An efficient suction apparatus should always be at hand, as it will prove useful in keeping the wound dry and so preventing any blood or secretions from being drawn into the lower air passages. If the operation is properly conducted scarcely a drop of blood should enter the trachea. I originally made use of a median incision, but on account of the possibility of fistula formation necessitating a subsequent plastic operation a flap is now employed. The incision which gives an adequate approach and which allows of speedy and permanent covering in of the wound in the larynx is fashioned, if the disease mainly occupies the left side, in the form of a mark of interrogation, and a reverse interrogation mark if it occupies the right side. This incision allows of a flap of skin being turned aside from the larynx while the lower vertical portion is employed for the tracheotomy. By carrying the upper limb as a horizontal extension to the left or the right sufficient room can be obtained to expose when necessary the whole larynx. The transverse part of the incision should correspond in level to the lower border of the hyoid bone; the limit of the outward curve should reach as far as the posterior border of the thyroid cartilage at about the centre of that border. The descending limit of the curve reaches the middle line at the lower border of the cricoid cartilage and is then extended vertically downwards as far as the suprasternal notch. The skin is now reflected and all bleeding points secured. The next step depends

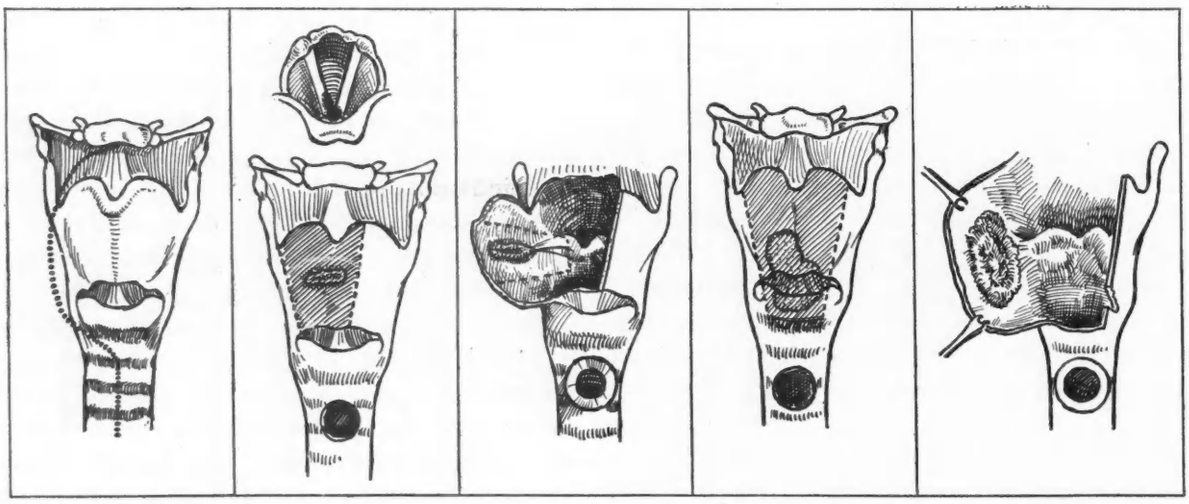

Fig. 1 Fig. 2 Fig. 3 Fig. 4 Fig. 5

Fig. 1.—Preliminary incision in a case of cancer affecting mainly the right vocal cord. All the drawings are semidiagrammatic. **Fig. 2.**—The extent of cartilage removed in a growth spreading from the right cord to the left across the anterior commissure. **Fig. 3.**—The cartilage has been removed and the mobilized soft tissues are pulled aside to expose the tumour. **Fig. 4.**—The amount of cartilage (thyroid and cricoid) which must be removed in an extensive growth, which has spread to the subglottic region. **Fig. 5.**—A further stage in the removal of a subglottic growth, as indicated in Fig. 4.

considerably on the extent of the tumour. This particular operation has been decided on because the disease has crossed the middle line anteriorily, or is mainly a growth of the anterior commissure, or has extended into the subglottic region. The growth, moreover, may be subglottic as well as bilateral, or it may have passed upwards so as to implicate the ventricular band. A large part of one thyroid ala and a portion of the opposite ala must be removed. The prelaryngeal muscles on the side chiefly affected should be turned aside; if they are merely retracted the surgeon is liable to be hampered by an inadequate view of the operative field. The muscles on the less affected side can be retracted. The whole of the thyroid cartilage on the side chiefly involved, with the exception of the posterior border and cornua, must be exposed, and as the growth has extended up to, or crossed, the anterior commissure, the anterior border of the thyroid and a portion of the opposite ala should be laid bare, the periosteum being carefully preserved. The central portion of the thyro-hyoid membrane (conus elasticus) is exposed to view, a keen look out being kept for any enlargement of the gland situated on this membrane, which is sometimes involved in intrinsic cancer. If the gland is discovered it should be removed, preferably with the endothermy knife, and preserved for subsequent microscopic examination. In order to describe better the further steps of the operation it will be convenient to imagine that it is being performed on a patient in whom the anterior half of the right cord is involved, the growth having crossed the middle line so as to implicate the anterior extremity only of the left cord. In such a case the whole of the anterior three-fourths of the right thyroid ala and the anterior third of the left should be removed. The whole depth of the cartilage may be resected, or the extreme upper margin can be left. Complete removal is preferable, as when the larynx is open a better exposure of the tumour is obtained, and it is questionable if retention of the upper margin of the thyroid cartilage is of any importance in maintaining the laryngeal thoroughfare. Punch forceps will be found most useful in removing the cartilage, and it is best to begin above. A separator is placed between the internal perichondrium and the thyroid ala and separation effected over a limited area. Cartilage is nibbled away in a downward direction, preceded always by further separation of the perichondrium and soft tissues. With a suitable pair of forceps sufficient cartilage or bone can be rapidly bitten away. If during this procedure any suspicious cartilage is encountered it should be kept for microscopic examination, and an endothermy separator may with advantage be substituted for cold steel. It must be admitted that in certain cases where before operation no suspicion of such a complication has been aroused the thyroid cartilage is found to have been penetrated by the tumour. At an earlier stage the internal perichondrium and the cartilage lying in immediate contact may be involved and the extension of the disease would not be recognized when the outer surface of the cartilage is examined. Such a condition would reveal itself when the cartilage is removed in the operation under consideration, and its recognition would lead to early steps being taken (diathermy or the actual cautery) to sterilize the region before considering the further planning of the operation. It may be stated here that strictly local involvement of the cartilage need not necessarily indicate total extirpation of the larynx, but if it is extensively diseased there should be no hesitation in carrying out the major operation. Sufficient cartilage having been removed, the time has now arrived to introduce a tracheotomy tube. It will be necessary in nearly all cases to divide the isthmus of the thyroid. After division a ligature should be tightly applied close to each cut surface. I am not certain that the preliminary injection of a solution of cocaine before opening the trachea is a sound procedure, as the cough which is induced on opening the wind-pipe helps to remove any secretion lying in the air passages.

A disk, which will include portions of the 2nd, 3rd and 4th rings, should be cut from the anterior wall of the trachea. If the cancer is thought to be subglottic the opening in the trachea should be made at a slightly lower level. At this stage some further information may be obtained by inserting a small laryngeal mirror into the tracheal opening. By using a frontal reflector a good view is obtained of the region of the anterior commissure and a subglottic extension may be discovered. In the next step of the operation I always insert through the tracheotomy opening a small sponge with a

thread attached, cut so as effectively to plug that portion of the respiratory tube corresponding to the lowest part of the larynx and uppermost segment of the trachea. This prevents the descent of any blood when the larynx is opened. The tracheotomy tube is inserted, and through it the anæsthetic is administered. The time has now arrived for opening into the cavity of the larynx. In many ways this is the most important step in the whole operation. It is essential that this entry should be made through healthy tissue, at a distance, if possible, of a half to three-quarters of an inch from the margin of the tumour. In thyro-fissure the thyroid cartilage is divided in the middle line, the proximity of the section to the tumour depending entirely on the nearness of approach of the latter to the anterior commissure. In my imaginary patient the growth has spread to the anterior extremity of the left cord, therefore the larynx should be entered well to the left side of the middle line. In some cases, however, it may be deemed advisable to open the cavity of the larynx at or near the level of the upper border of the thyroid cartilage. In this way the operator can more easily keep clear of the tumour. Another method is to get an assistant to bulge out the healthy part of the larynx above the margin of the tumour with a rigid angled instrument inserted through a directoscope. This instrument is palpated from the outside and the incision can be made over it. An endothermy knife may be used in opening into the larynx and in carrying out the rest of the excision. It should certainly be employed when there is thought to be any risk of cutting into the tumour. It might be quite feasible with an endothermy needle bent at a right angle to open the larynx from within outwards, employing a directoscope. There would be no hæmorrhages if this procedure were adopted, and therefore the incision could be made with great accuracy under direct vision. Once the larynx is opened, all that is necessary to obtain a general view of its interior is to apply traction to the cut edges of the wound everting the portion on which the tumour is situated when it can be viewed as in laryngo-fissure *en face*. The advantage of preliminary removal of the overlying cartilage is that the part to be removed is mobilized. Systematic excision can now be carried out, keeping the surface and edge of the tumour in view all through the remainder of the operation. The exposure is quite as good as that obtained in thyro-fissure, and in a suitable case there is no excuse for not excising an adequate amount of normal tissue all round the tumour. It is also extremely easy to ascertain the lower limits of the growth. If it is found to extend into the subglottic region it is best to resect the anterior part of the cricoid cartilage. When this has been accomplished the tumour can be traced downwards and removed, a portion of the crico-thyroid membrane being taken away, or a segment of the cricoid with the tumour lying on its posterior aspect may be removed in one piece. If the tumour occupies the anterior commissure a slight modification of the operation is all that is necessary. In certain cases, when the tumour cannot be removed completely, extirpation of the larynx may be avoided by treating any remaining portion of the disease by the insertion of radium.

The after-treatment does not differ in any respect from that of laryngo-fissure, except that it is generally advisable to leave the tracheotomy tube in position until healing has occurred. In a limited number of cases a permanent tube will have to be worn, but it is remarkable to find, so long as the mucous membrane covering the arytenoids, interarytenoid space and anterior aspect of the posterior ring of the cricoid cartilage remains, that the space after healing has taken place is adequate for breathing purposes and a tolerable voice may be retained.

The Julius Rosenwald Fund in cooperation with other agencies has succeeded in making good medical care more available to people of small means. The Trustees at a recent meeting expressed their continued interest in medical economics and their desire that programs in this field be carried forward effectively. Voluntary insurance to lower the cost of hospital care—group hospitalization—has doubled, now covering some 350,000 persons with plans being established monthly in new communities. A recent publication of the Fund described new plans for securing general medical care on a group payment or insurance basis, conducted in many parts of the United States by industrial agencies, cooperative societies, groups of physicians and medical societies and benefiting several million people. The Fund will continue to assist in the extension of the insurance principle in relation to hospital service, in scientific studies concerning the cost and the organization of medical care and in the education of qualified personnel for studying and administering hospitals and clinics.

A CLINICAL AND ANATOMICAL STUDY OF SCIATICA*

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GREAT confusion still exists regarding the etiology of chronic sciatica. Before the advent of the x-rays sciatica was considered to be an inflammation of the nerve sheath, and was treated largely by drugs and physiotherapy. Arsenic, mercury, potassium iodide, iron, salicylates and many other remedies were in use. It was not thought to be due to pressure or to have a bony origin. With the coming of x-rays the hip-joint and, later on, the spine were thought to be sources of the pain, which was attributed largely to arthritis with "lipping". In 1905 Goldthwait and Osgood may be said to have discovered the sacro-iliac joint, with its tendency to strain and relaxation. Thus, following arthritis, joint strain, especially of the sacro-iliac joint, was added to the etiology of sciatica. The sciatic pain and backache were thought to be referred in some way from sacro-iliac and other lesions, much as a pain at the knee may be caused by disease of the hip-joint. In 1925 Danforth and Wilson pointed out the importance of the lumbo-sacral foramen as a source of sciatic pain. Smith-Petersen, of Boston, was relieving many cases of sciatica by fusing the sacro-iliac joint, an operation which he had devised. To many, however, it was difficult to explain the undoubted and often immediate success of this operation before fusion could have occurred, perhaps with little or no x-ray evidence of a lesion of any kind. Heyman[1] recently reported cases in which pain was relieved by simple exposure of the sacro-iliac joint, a procedure involving considerable stripping of the gluteal muscles from the ilium, but without going on to fusion. Ober,[2, 3] of Boston, was quick to realize the significance of this. He has proclaimed that the success of sacro-iliac fusion lies often in the muscular stripping and the consequent relief of tension. The tension, he maintains, is largely concentrated in the ilio-tibial band. As a remedy he cuts this band. His results have been good.

The writer was stimulated to a study of sciatica by a personal communication from Prof. S. E. Whitnall, of the University of Bristol, late Professor of Anatomy at McGill University. Professor Whitnall told of a case of chronic sciatica which had been relieved by a Bristol surgeon. The surgeon had cut down on the sciatic nerve in the buttock, and had found a band pressing on the nerve near the sacro-sciatic foramen. The band was cut with relief of the sciatica. It was thought that the band in question was the inferior margin of the gluteus medius muscle. Professor Whitnall suggested to the writer that a study of this band might be made with profit in the anatomical laboratory of McGill. With the kind cooperation of Prof. J. S. Baxter, of the Department of Anatomy of McGill University, several bodies were examined, and the following general observations made.

ANATOMY

The muscles of the gluteal region are arranged in three well-defined functional units placed on successive planes. The deep group is comprised of the gluteus minimus, piriformis, gemelli, and obturator internus, in that order from above downwards. The fascial sheaths investing the members of this deep group are continuous one with another and blend also with the fascial tube investing the emerging piriformis and great sciatic nerve. This fascial tube is really a prolongation of the parietal pelvic fascia drawn laterally through the great sciatic notch by the structures mentioned, namely, the piriformis and the great sciatic nerve.

There are two points at which the fascial coverings of these structures are thickened: (*a*) where the upper border of the piriformis is contiguous to the lower edges of the gluteus medius and minimus; and (*b*) at the lower

* From the Orthopædic Department of Montreal General Hospital.

border of the piriformis immediately lateral to the great sciatic. It has been noted that where the intervals between these related structures are greater than normal there is more likely to be greater thickening of these fasciæ, even to the extent of definite bands. Of particular significance is the space between the piriformis and the gemelli. (Figs. 1, 2 and 3).

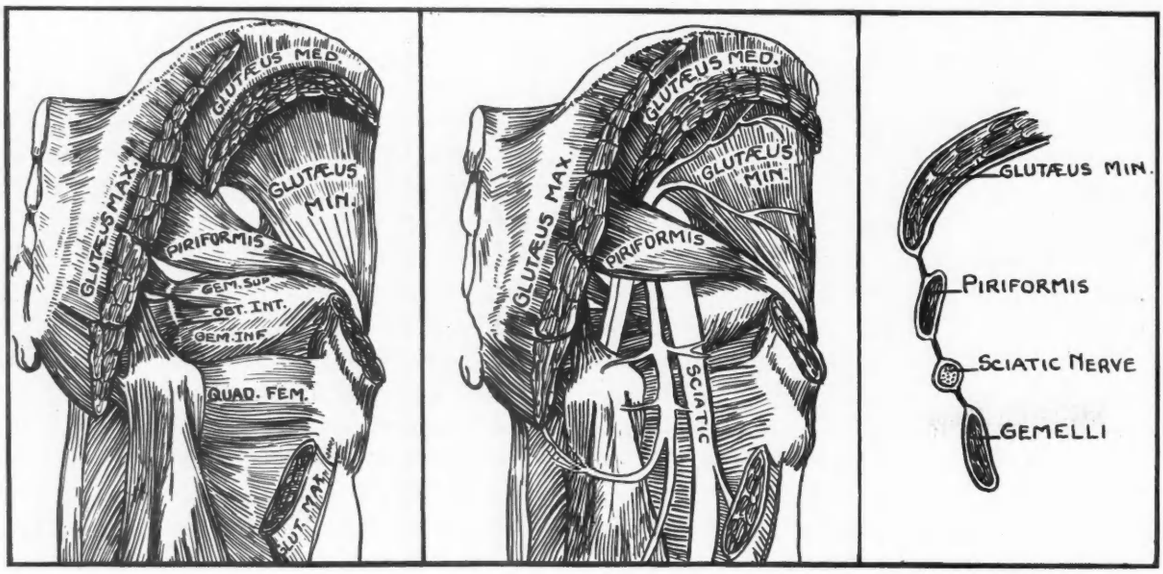

Fig. 1 Fig. 2 Fig. 3

With these findings in mind the following cases were collected and dealt with as described.

CASE REPORTS

CASE 1

D.M., male, aged 35. Pain developed in the left sacro-iliac region on October 3, 1934, while the patient was doing heavy work. The pain radiated through the buttock and along the thigh to the calf and ankle and has persisted.

The man was well developed and well nourished, and stood with his weight on the right foot, with a marked tilt to the pelvis. Marked limitation of motion of spine; marked tenderness over the left sacro-iliac region and over the course of the left sciatic nerve; marked limitation of motion on left straight leg-raising. X-ray showed well-marked condensation of bone about the right sacro-iliac joint, which would indicate arthritis in this area. The articulation on the right side between the fifth lumbar and first sacral vertebræ showed changes indicating arthritis, also narrowing of joint space in the lateral articulations; no narrowing of intervertebral disc. There was evidence of lipping of the lower three lumbar vertebræ and a spina bifida of the lower sacral vertebræ. The lumbo-sacral curve was rather flat. The ilio-tibial bands were not examined before operation.

As chronic arthritis, past or present, was indicated in the skiagrams of this patient, a diligent search was made for foci of infection. The tonsils were removed and the teeth were put in good condition. The Wassermann test was negative; genito-urinary examination was negative. The man was baked, his pelvis strapped; he was put in a plaster spica; and he was treated with salicylates. His sciatica with its attendant scoliosis stubbornly persisted.

On April 9, 1935, manipulation of the spine and a plaster spica under anæsthesia relieved the patient of his pain only temporarily, and by the end of August, 1935, the question of sacro-iliac fusion was considered.

On September 11th, under spinal anæsthesia, the sciatic nerve was explored at the sacro-sciatic foramen. An incision was made across the left buttock, beginning at the upper margin of the great trochanter and extending mesially and upwards. The muscle fibres of the gluteus maximus were separated by blunt dissection and the sciatic nerve was exposed, emerging from below the pyriformis. Under the latter muscle a band was felt, which, it was thought, might possibly be pressing on the sciatic nerve. This band, which did not resemble an adhesion, was cut. No attempt was made to lift the sciatic nerve from its bed, but the exploring finger, after cutting the band, was able to follow the nerve into the foramen without further obstruction. On recovering from the effects of the anæsthetic, the patient announced that his pain was gone. His relief has been permanent to date (June, 1936). His sciatic scoliosis disappeared rapidly.

CASE 2

E.D., male, carpenter, aged 48. Pain low in the back radiating down the posterior surface of the right thigh and leg to the heel, present since December, 1934. No trauma. The pain was present even in bed, varied in intensity, and was worse when walking. He could relieve it momentarily by bending forward with the right knee flexed. He was quite unable to lie on his right side. Pain, from being a dull ache, had recently assumed a burning character.

The patient was a tall, spare man. There was a history of gonorrhœa in 1918 and of meningitis (?) in 1927. Teeth, (mostly extracted); no abscesses. The tonsils were small, with prominent follicles and cheesy particles.

No tenderness over the lumbar spine. Normal movement in hips and spine. No atrophy of the muscles of the right leg. Tenderness behind the right thigh and calf and to the outer side of the right ankle. Reflexes normal, except for diminished right ankle jerk. No evidence of cord tumour. Straight leg raising was possible to 80°, when pain was produced at the back of the right calf. Tenderness was present here as well as over the right sacro-sciatic foramen in the buttock, where it was acute. There was slight tenderness on pressure also over left sciatic notch, which the patient stated he had noticed for the last two days following a long automobile ride. Straight leg-raising (left) could be carried to 90° with-

out pain. The ilio-tibial bands were not examined before operation.

X-ray showed condensation of the bone about both sacro-iliac joints, which suggested arthritis. There was spina bifida of the first sacral segment. The lower border of the fifth lumbar vertebra showed condensation and lipping suggestive of osteo-arthritis in this area. The lateral view showed a rather flat lumbosacral curve.

The patient's blood sugar tolerance was found to be impaired, and for a week he was given special diet and insulin by the Department of Metabolism. His blood Wassermann test was negative, as also the colloidal gold curve of the spinal fluid.

Operation, October 15, 1935. An incision was made transversely across the right buttock, on this occasion at a somewhat lower level, *i.e.*, about an inch below the level of the great trochanter. On going through the fibres of the gluteus maximus an ill-defined broad band of fibrous tissue was found running transversely across the nerve at the lower border of the piriformis. It was on the lateral side of the nerve and was thin enough to be torn by the exploring finger, which could then follow the nerve freely into the foramen. The muscle was closed by catgut, the skin by silkworm. The pain persisted for some time after operation, but in about two weeks he was discharged with nothing but slight tenderness over the right sciatic notch. Up to the present (April, 1936) he has remained free of pain.

CASE 3

J.P., aged 51, male.

On October 7, 1935, this man came to the clinic complaining of acute pain down right leg, which began as an acute pain in the back on July 13, 1935, when working; no special trauma. A doctor strapped his back, and after 16 days the pain subsided. Soon after the pain reappeared, now radiating down to the buttock, thigh and ankle. It was fairly constant; walking relieved it somewhat; standing increased its severity. He could lie in bed only on the left side with the right knee flexed; otherwise the pain was severe. No pain in the left leg or any other joints. The right foot felt numb. For the past 21 years the man had worked as fireman in Canadian National Railways and other plants. He had not worked since the onset on account of the severity of the pain.

The patient was a well developed, rather heavy Italian. Both tonsils were submerged and infected, and there was definite cloudiness in the right antrum. No evidence of apical infection in the teeth, which, however, were dirty and ill-kept. Sciatic scoliosis was present; concave to the right. No vertebral pain or tenderness. There was slight tenderness on pressure over both sacro-iliac joints. Tenderness over the sacro-sciatic foramen was marked, and to a less extent over the back of the right leg. Straight leg-raising (right) was completely inhibited by pain at 50° of flexion. The ilio-tibial bands showed moderate tightening on the right side; the left was probably normal. The Wassermann test was negative; examination of spinal fluid, together with the colloidal gold test, negative. Blood sugar tolerance normal. X-ray showed very marked lipping at the margins of the lower lumbar vertebræ, and there was evidence of arthritic changes about both sacro-iliac joints, particularly the right. There was lipping about both hip joints; no evidence of bone destruction, and no loss of joint spacing. The lateral view showed a rather flat lumbo-sacral curve. Ankle jerk was absent on the right side, and there was an indefinite area of diminished sensation to pin-pricking along the outer border of the right foot. There was vague hyperæsthesia up the back of the right calf and thigh.

Operation, October 17, 1935, under spinal anæsthesia. The sciatic nerve was exposed at the lower border of the piriformis muscle. A band of fibrous tissue was encountered running transversely across the nerve as it emerged from below this muscle. This band was in close contact with the nerve and was cut, being too tough to be torn. After this the exploring finger could follow the nerve up into the foramen without further obstruction. Usual closure. In three weeks' time the patient was getting about without pain, but this returned when lying in bed. In another week he was discharged, with nothing but a feeling of numbness over the right ankle. Three months later he reported that he could do heavy work and only had occasional pain on lifting heavy weights.

CASE 4

J.N., male, waiter, aged 32, complained of pain in the back radiating down left leg since April, 1935. He had had a primary chancre two years ago. No gonorrhœa. Married 9 years. Sciatic pain followed a sore throat.

The patient was a slight but healthy-looking male. The tonsils were large and injected; the tonsillar glands were large, firm but not tender; the inguinals firm and fibrotic, not tender. The left thigh and leg showed marked muscular atrophy. The pelvis was tilted; lumbar scoliosis with convexity toward the right. Limitation of motion of spine in all directions. Marked tenderness over left sacro-iliac joint and left sciatic nerve. Lumbar lordosis reversed. The urine was negative; Wassermann test negative. X-ray showed arthritic changes (lipping) about the sacro-iliac joints and the lower lumbar vertebræ; no bone destruction; no loss of joint spacing. The lateral view showed a rather flat lumbosacral lordosis.

On account of the sciatic scoliosis and reversed lumbar curve the man was admitted to the wards on July 15, 1935. Under an anæsthetic the adhesions were broken down, the sciatic nerve was stretched, the lumbar curve was re-established and a plaster of Paris spica applied. He was discharged ten days later in spica. A few weeks later, on removal of the cast, his pains returned. He was readmitted on September 9, 1935. On this occasion his spinal fluid, as well as his blood, showed a completely negative Wassermann test. In addition, the spinal fluid showed a negative colloidal gold curve. An abscessed tooth was removed. He was examined by the Neurological Department and no evidence of a cord lesion was found. The diagnosis of left sciatic neuritis was confirmed. There was evidence of diminished sugar tolerance, which was treated by the Metabolism Department, insulin and an appropriate diet being used.

The patient on the second admission had relapsed to much the same as his previous condition. He still showed the familiar sciatic scoliosis with flattening of the lumbar curve, limitation of straight leg-raising, tenderness over left sacro-iliac joint, also over the sciatic nerve at the sacro-sciatic notch and down the leg. On September 12, 1935, his spine was manipulated for the second time under gas and oxygen. Following this the patient had no pain for several weeks. Three weeks after manipulation his spica was bivalved and daily baking and massage begun. The pain returned.

October 25, 1935. Marked tenderness over sacro-sciatic foramen existed on the left side. There was a certain amount of flexion contracture of both hips; no hyperextension was possible. The ilio-tibial band seemed to be more tense on the good, or right, side than it was on the left. As the patient evidently was not cured the left sciatic nerve was explored on October 29, 1935, in the usual way. A band was found mesial to the nerve, but apparently not touching it. This band was removed.

November 18th. The symptoms were greatly improved, there being only slight pain above the outer ankle. On discharge, November 22nd, there was no tenderness over the course of the sciatic nerve. Straight leg-raising to 90° was possible on both sides without pain, February 28, 1936. He was working in a restaurant 12 hours a day, standing all the time; no pain save an occasional slight soreness over the lateral aspect of the left ankle; no tenderness over the lumbo-sacral or sacro-iliac joints, sacro-sciatic foramen or superior gluteal nerve. Straight leg-raising on both sides was free, equal and painless.

CASE 5

L.Y., male, aged 56. His complaints were (1) pains in the muscles of the left hip for 10 months; (2) œdema of the feet and legs for five months. There was no history of trauma to the hip. The pain was dull and constant, aggravated by movement and by straight leg-raising; no pain in any other part of the body. Damp weather intensified the pain. The patient was unable to stoop or sit down without pain. The diagnosis in the medical ward was (1) sciatic neuritis, left; (2) arterio-sclerotic heart disease with decompensation; (3) generalized arteriosclerosis; dental caries with marked oral sepsis; lues (latent?); hæmorrhoids.

Examination on November 4, 1935. The patient stood and walked in a stooping position. There was marked lumbar lordosis, at a higher level than normal. The whole spine, dorsal and lumbar, was largely fixed by chronic arthritis. There was flexion contracture of the left hip, apparently due to the soft parts. There was slight flexion contracture in the right hip. The left ilio-tibial band could not be examined properly owing to flexion contracture of the left hip. It was probably quite tight. The right was probably normal or slightly contracted. There was no pain in the back itself. X-ray showed marked arthritic changes in the sacro-iliac joints and margins of the lower lumbar vertebræ; no arthritic changes were seen in the hip joints. In the lateral view lumbar lipping was seen. The main tenderness seemed to be at the sacro-sciatic foramen. Some sacro-iliac tenderness was present. Crest compression and separation probably negative. The case was considered to be sciatica associated with chronic arthritis, which in turn had brought on postural changes. The patient, after treatment on the medical side, was transferred to the Orthopædic Service on November 19, 1935.

Operation, December 3rd, exploration of the sciatic nerve. Only diffuse fibrous tissue bands were found crossing the nerve; no mark seen on the nerve trunk. The wound healed uneventfully.

December 20th. The pain continued in the left buttock and the region of sacro-sciatic notch. The man was up and about and had developed œdema of the ankles. To remain in bed. January 2nd, 1936. The pain over the foramen was gone, the pain and tenderness in the region of the left tuber ischii, which radiated down the thigh and had been present for some weeks was not affected by the operation. Some pain and tenderness in lower lumbar region, probably arthritic. The pain was much increased when in bed. Strapping of the pelvis was of little service.

January 27th.—Tenderness over the sacro-sciatic foramen had not returned. The plaster spica had not been of service. Pain persisted in the region of the left tuber ischii and down the thigh. Allowed to go home.

CASE 6

Miss G. S., aged 29, stenographer, complained of pain on the right side of the lower back, in the right buttock, and down the right thigh, almost to knee, for five years off and on. The pain was not particularly related to standing, sitting or lying down, but appeared at any time. Occasionally it affected the left side. Probably there was no trauma at the onset. No pain was complained of in other parts of the body. The pain was present in recumbency, on movement, and mostly in the right buttock.

Examination, November 20, 1935. The patient was sparely built and healthy. Tenderness over right sacro-sciatic foramen. No pathological reflexes. No sensory changes. Lumbar lordosis was exaggerated, but not fixed, and not obliterated by forward flexion. Marked tenderness, but no pain over the right sacro-iliac joint. The feet were moderately pronated. Crest compression negative: crest separation, right, positive, left, negative. The right ilio-tibial band was markedly contracted, the left normal. Hip movements were normal. Straight leg-raising was positive or negative according to the severity of the pain. X-ray showed some sacro-iliac lipping, more marked on left side. Lateral x-rays were negative, save for an increased lumbo-sacral curve. Strapping the pelvis did not give relief. Exploration of the right sciatic nerve was advised on account of chronic tenderness over sciatic nerve at the sacro-sciatic foramen.

November 28th.—The right sciatic nerve was cut down upon. Several thin, tough bands were found to cover the nerve quite closely for about one inch under the piriformis muscle. These were cut or torn. Uneventful healing.

There was no pain for nearly two months after operation, when it gradually returned, similar in character but to a less extent. Examination, January 22, 1936, showed lumbo-sacral and right sacro-iliac tenderness. The pain disappeared suddenly and was not constant. There was no return of tenderness over the sciatic nerve. New pain was complained of on the outer side of the thigh. As the right ilio-tibial band was known to be tight, it was advised that it be cut. This was done on February 4, 1936, under general anæsthesia. Incision from below antero-superior spine, mesially, to above and beyond great trochanter. The ilio-tibial band seemed not only tight but thick and the severed ends separated widely. The pain in the back and buttock and down the leg disappeared soon and had not returned to date (March 21, 1936). The patient volunteered the statement that she felt she was standing straighter as a result of the last operation. She complained of a slight feeling of stiffness between her shoulder blades, this probably being due to a change of posture. No return of pain to end of May, 1936.

CASE 7

J.G., male, aged 27, was admitted to the Jewish General Hospital on November 1, 1935, in the service of Dr. S. E. Goldman, with a history of pain in the right buttock, radiating down the right leg, of about 9 years' duration, with intermissions. For the past six months his attacks had been increasing in severity until finally he reached the stage where he took to bed.

The physical findings were as follows. He stood with difficulty and marked pain; right-sided scoliosis; restriction of all movements; reversed lumbar curve; straight-leg raising markedly restricted. Marked tenderness on pressure over the right sacro-sciatic foramen. Skiagrams of the spine and sacro-iliac joints were negative. His tonsils were removed. A non-specific prostatitis was cleared up, but no other positive source of infection was found. On November 21st the spine and affected leg were manipulated and a spica applied. This gave only partial relief for a short period.

Examination on March 23rd showed his condition to be essentially the same as on the previous examination. At this time it was noted that there was a marked tightness of the ilio-tibial band on the right side (Ober's sign). He had also marked tenderness over the upper aspect of the right buttock somewhat above the sacro-sciatic foramen. An exploration of the sciatic nerve was decided upon and carried out by Dr. Goldman on March 27, 1936, under general anæsthesia. The right ilio-tibial band, even under ether, remained contracted. The sciatic nerve was exposed at its emergence from under the piriformis. A blunt hook was passed beneath the nerve and the nerve lifted from its bed. No abnormalities were noted; no adhesions or bands, fibrous or muscular, could be seen or felt and the nerve appeared normal. A portion of the gluteus maximus was stripped by blunt dissection from its origin on the posterior superior spine and forward along the crest of the ilium for a distance of about two inches. It was also freed somewhat at its lower end where attached to the ilio-tibial band. Subcutaneous closure was with gut; of the skin, with silkworm gut. Pain disappeared the same day and the patient remained free of it for 6 weeks. Ilio-tibial band cut May, 1936, with good results to date (June).

CASE 8

Mrs. A.C. The patient was a large woman of neurotic type, complaining of backache and left sciatica for the last two and one-half years.

Examination, March 24, 1936.—Pain and marked tenderness in left sacro-iliac region, buttock and leg. Lateral movements of the spine were very fair; forward and backward flexion were restricted and accompanied by the usual pain. Left limp. No gluteal atrophy; no atrophy of the muscles of the buttock. The ilio-tibial bands were normal on both sides. X-rays showed somewhat exaggerated lordosis. This was not obvious when the patient was standing. X-ray also showed marked lipping of the right sacro-iliac joint. The pain persisted in bed. A corset had not helped. She kept her left knee flexed in the erect position. Tenderness was present over the left sacro-sciatic foramen.

Operation, April 1, 1936, Woman's General Hospital. The usual exposure of the left sciatic nerve through the gluteus maximus. No definite band could be made out on nerve trunk. Some resistance was felt to the finger exploring upwards along the nerve from fibrous tissue, which was easily torn. There was no evidence of pressure by overlying muscles. The incision in the gluteus maximus was prolonged laterally to the femur, where fibrous intersecting bands were cut by scissors to relieve muscle tension at its insertion.

April 8th. The characteristic pain in left sacro-iliac and back of leg was now gone although the wound presented some soreness. A new pain had appeared in front of the left hip, apparently associated with tenderness in the left lower quadrant (adhesions following old abdominal operation?).

April 27th. The patient was sent to the Montreal Convalescent Hospital. Backache and sciatica were absent except when she had been up and about for a long time, when it was felt to a slight extent. The patient, although distinctly of the complaining type, was pleased with the result. Pain has not returned to end of May, 1936.

CASE 9

W.M., aged 39, complained of acute pain in back and down left leg since early in October, 1935. Tenderness was present in the left sacro-iliac region and over the left sacro-sciatic foramen. X-ray showed lipping about the right sacro-iliac joint and lower lumbar vertebræ. The tonsils were badly infected and were removed on December 7, 1935. No other infective focus was found. A month later he was no better. The principal pain and tenderness were now at the lower margin of the buttock rather than over the foramen.

February 7, 1936. The sciatic nerve was explored and found to be surrounded by diffuse, rather delicate, fibrous bands, not giving any visible signs of pressure. These were cut. No muscular pressure was found. No sciatica occurred for a month, when it returned. The left ilio-tibial band showed some, but not marked, tightening; on the right side it was perhaps slightly contracted.

March 24, 1936. Under local anæsthesia the left ilio-tibial band was cut. The pain, which was felt slightly for a couple of weeks when he coughed, finally disappeared completely. He was last seen on April 17, 1936.

Analysis.—No nerve pressure was found. Some relief of tension, resulting in temporary freedom from pain, was apparently afforded by the approach through the gluteus maximus. Possibly the activity of this muscle was for a time inhibited as a result of the operation. Evidently the relief of tension was not sufficient. Cutting the tight ilio-tibial band brought about a cure.

CASE 10

J.M., male, aged 49, laborer, in October, 1934, began to have pain in the right hip running down the posterior side of the leg to the ankle. By the end of December, 1934, pain was so severe that he was admitted to the Medical Service of the Montreal General Hospital. On examination he was found to have infection about the roots of several teeth, two being subsequently extracted. X-ray indicated sinusitis, frontal and maxillary. The throat and tonsils seemed healthy. The right leg showed the usual limitation of forward flexion with the knee extended with definite tenderness over the right sacro-sciatic notch. There was flattening of the right buttock. Hip movements were free, and x-ray showed evidence of lipping about the right hip-joint, at the margins of lumbar vertebræ, and about the sacro-iliac joints, especially the left. Diagnosis.—Chronic neuritis of the sciatic nerve. After a month he was discharged, but never remained completely well. Exposure to cold and change of weather brought on pains in lower back and both legs.

The patient was readmitted on October 8, 1935. His spine was straight. He showed tenderness to pressure over both sacro-sciatic notches, but more marked on the right, also over the back of both calves. Straight leg-raising was limited on right side to 80°. The hip joints were free. The ilio-tibial bands were not abnormally tight on either side. Slight sensory and reflex changes in the right leg. Examinations of the various systems were made with negative results, save as to maxillary sinusitis.

On October 18, 1935, the right sciatic nerve was explored in the buttock. A tough band of fibrous tissue was encountered running transversely across the nerve and apparently capable of exerting pressure on it. No sign of pressure was seen on the nerve trunk itself. The band was connected to bone at its lateral end, and mesial to the nerve it blended with the fibro-fatty tissue of the region and was lost. The band was too dense to be torn by the fingers and was excised in two places. No nerve pressure from adjacent muscles was made out.

The patient was deported to Rumania not long after and has been lost sight of. The final result is uncertain but the man was free from pain on exit about 3 weeks after operation.

CONCLUSIONS

The first four of these cases were apparently permanently cured by exploration of the nerve with section of any bands found. In no case was there seen any evidence of irritation of the nerve trunk. Two cases (2 and 3) showed bands that might conceivably have caused direct pressure on the nerve. These four successful cases were of the flat back type. The ilio-tibial bands, examined before operation, showed marked shortening in cases 7 and 9 and perhaps in case 4. Cases 5, 6, and 9, subjected to nerve exploration, were not relieved by this operation. The first two showed increased lumbar lordosis and a tight ilio-tibial band on the affected side. Patients 6 and 9 were finally completely relieved by a second operation, *i.e.*, section of the ilio-tibial band.

The question then arises as to what brought about the relief of symptoms in these cases. It would appear to the writer that relief of muscular tension played a part. Is this increased tension, as for example caused by a tight ilio-tibial band, to be considered as secondary to a chronic arthritis in neighbouring joints, or is it due to inflammatory changes

in the muscles themselves, with secondary shortening? Such a reaction has been reported by Ober.[2] Again, does this increased muscular tension cause pain by direct pressure on the nerve trunk, or does it act indirectly by causing joint strain or postural changes? Further study is necessary.

The writer wishes to acknowledge with grateful thanks the kind assistance and cooperation of Professor J. S. Baxter of the Department of Anatomy McGill University and the prosector Mr. Muir; as also of two of his assistants in the Orthopædic Department of the Montreal General Hospital, Dr. S. E. Goldman and Dr. J. G. Shannon.

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PRIMARY SARCOMA OF THE BREAST*

(WITH A REPORT OF FOUR CASES)

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JUST as philosophy, through the ages, has suffered restrictions of its scope by the branching off of the delimited sciences, so "sarcoma" of the breast (a term which once designated almost any tumour not carcinoma) has become an active and positive factor in the field of breast pathology and of pathology in general. Sarcoma now has definite subdivisions, each of which can usually be said to *be* something, rather than *not be* something else. It is necessary that we say "usually", for Ewing tells us that even the spindle-cell structure which has been so universally associated with the mesoderm is not necessarily sarcoma. Some "spindle-cell sarcomata" prove by their metastases to be atypical carcinomata. They are usually quite malignant. And so, to quote once again Ewing's much quoted paragraph,[3] "When one excludes from the group of mammary sarcomata the adenosarcomas, the malignant forms of mixed tumours containing cartilage, bone, mucoid, or fat tissue, and certain malignant round- spindle- and giant-cell alveolar pseudo-sarcomas, which are atypical carcinomas, there is little remaining of a once formidable group of mammary neoplasms".

A sarcoma, say Deaver and McFarland,[2] is a tumour arising through vegetative activity of connective tissue. It is characterized by rapidity of cellular multiplication in excess of cellular differentiation. Muir[6] adds that sarcomata are characterized by a rich blood-vascular system, the channels of which are lined by a single layer of cells resting directly on the tumour cells. This characteristic leads to bleeding and metastases through the medium of the general circulatory system. Lymph channels are affected to any considerable extent only in lymphosarcoma and melanotic sarcoma. These, of course, are of little concern in the particular topic for discussion.

Classification.—A purely mesodermic tumour is exceedingly rare in the breast. While exact figures are not given, all authors writing on the subject point out that a sarcoma with no epithelial element present is most unusual. Classification of these tumours, therefore, is based upon the morphology of the predominant cell type. Thus, we have described for us adenosarcomata and pure sarcomata. But the adulteration of the one does not alter its clinical course as compared with the other. Cheatle and Cutler say that the two are indistinguishable, save in the laboratory. So we shall continue our discussion of the two as one.

The majority of cases of sarcoma and adenosarcoma of the breast give a history of a previously existing "lump", not large, most of them probably being fibroadenomata. (Who can tell?) This "lump" will have been present for many months or years. Now fibro-adenomata contain all the specific elements of breast tissue. Hence, when the connective tissue commences to reproduce, following a new set of laws, the motivating force often induces hyperplasia of the epithelial elements as well. If not, the elements present may not be stifled but remain to appear in the ensuing malignant neoplasm. Absence of epi-

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thelial elements does not eliminate the fibroadenoma as a precursor, but it certainly points the arrow of probability to a different quarter.

When epithelial hyperplasia ensues, often a cystic type of sarcoma results — huge in size, multilobular, and possibly with a pedicle. When the epithelial cells fail to multiply a pure sarcoma is found. Whether pure or not, sarcomata may be classified according to cellularity and according to type of cell. Both to a limited degree may indicate the potential malignancy. The solid tumours are smaller than the cystic variety, are encapsulated, firm and elastic. Very cellular forms may be more homogeneous and dull grey in colour, while the less cellular are more elastic, less fleshy, and are not so disposed to ulceration, hæmorrhage, and necrosis, which are so frequent among those containing cysts.

All varieties of sarcoma show marked blood vessel growth and may develop myxomatous changes.

Another classification, necessarily microscopic, is by cell type. The common type is the so-called spindle-cell sarcoma, mutations of which are the round cell and polygonal cell. The latter two may be superimposed or may entirely replace the former. The round-cell sarcoma is the most malignant of all, but must be thoroughly studied to differentiate it from an atypical type of carcinoma. Cheatle and Cutler[1] make a strong point of this deceptive quality. Malignancy decreases through mixtures of round cells and spindle cells to its least degree in the less cellular spindle types.

Deaver and McFarland[2] note that any tumour with giant cells may be called giant-cell sarcoma. In their series of cases they found no giant cells unassociated with typical spindle-cell sarcoma, and are of the opinion that the giant-celled form is but a variant of the purer type.

A third system of classification is mentioned by Regan,[7] its basis being age. Type I appears in the young. There is slow growth and few symptoms develop. So well encapsulated are these tumours that they may be successfully and completely shelled out at times. Type II appears later in life. Regan thinks that it may be derived from the intra-canalicular type of fibroadenoma. It is soft, cellular, and polypoid, with a pronounced tendency to ulcerate the skin—this, of course, through pressure and not infiltration. Type III is the solid tumour that occurs in later life and shows early distant metastases.

Cheatle and Cutler became particularly interested in the determination of the particular part of the connective tissue which might be the point of origin of the malignant changes. They tried to determine this by means of elastic tissue stains, but found this particular tissue to be absent or refractive. They state that they are unable to identify the responsible part of the stroma.

Incidence.—In 1915 Geist and Wilensky[4] reported the presence of 435 cases of sarcoma of the breast in the literature. They found that 3.9 per cent of all breast tumours and 7.7 per cent of all malignant conditions of the breast were sarcoma. Cheatle and Cutler reported 3.9 per cent of consecutive breast tumours to be sarcoma, and noted that the percentage is higher in the older literature, for obvious reasons. Regan gives the Mount Sinai Hospital reports as 7 per cent of sarcoma in a series of breast lesions. He thinks that the percentage given at Washington University, St. Louis, (3 per cent) is a more plausible figure.

In 1932 Schreiner and Thibaudeau[8] found the total of 510 cases of sarcoma of the breast—an increase of 75 for the previous seventeen year period. Of these a considerable number, particularly among the older specimens, were probably wrongly diagnosed. They found 7.8 per cent of tumours of the breast to be of connective tissue origin. Their statistics further show that 0.9 per cent of all sarcomata are of the breast, and that 0.5 per cent of all malignant conditions of the breast can be proved to be sarcoma. Hertzler,[5] in 1933, reduced this last figure to 0.2 to 0.1 per cent of sarcoma in the cases of malignant tumour of the breast.

Age.—The age incidence varies from four months to eighty-eight years (Geist and Wilensky), but the usual figures are given as 9 to 76. Cheatle and Cutler state that the optimum age is 40 to 45, while the general age level, as observed by Regan, is 30 to 65.

Sex.—Ninety-eight per cent of cases are in the female, leaving but 2 per cent for the male sex (Regan).

Etiology.—Unknown! Trauma, lactation, sexual activity, heredity, one or all of these, may play a part, but to none can any considerable number of cases be definitely traced. Probably on trauma is the most blame laid, because a

previously inactive nodule is so frequent a precursor of later malignant change. Trauma is very easily presumed to be the trigger that sets off this unfortunate discharge. After all, what woman's breast has not had some trauma at some time? We consider that trauma is really too ubiquitous an occurrence to merit a place in the etiology.

As has been noted, the majority of these tumours commence in association with a long-existing nodule in the breast. There is sudden rapid growth, occupying but a few weeks or months, during the process of which a fair size is obtained. All the while the growth remains circumscribed, and is generally freely moveable beneath the skin and over the chest wall until extensive growth has resulted. Except in the cystic type growth is uniform. Seldom is there extension and invasion, save in old subjects and in those specimens devoid of definite encapsulation. Necrosis of the skin, when it occurs, is due to pressure and not as a rule to invasion of epidermis by the tumour itself. The nipple is not retracted, and seldom is there any discharge from it. Pain is a symptom in about one-third of the cases, and is considered to depend upon the rapidity of the growth in most instances.

Diagnosis.—Diagnosis is essentially clinical as well as pathological, and seldom is made before the breast is removed. The exact classification is obviously determinable only under the microscope. Cheatle and Cutler summarize the differential points in diagnosis by saying that size, circumscription, mobility, lobulation, elasticity, and history of rapid growth generally eliminate carcinoma and pure forms of fibroadenoma. Absence of adherence to the skin, of nipple retraction, and of palpable axillary glands throw strong doubt on carcinoma. However, let it be reiterated that an early diagnosis without section is practically impossible.

Treatment.—Radical removal with wide skin flaps—so wide that subsequent skin grafting is required—is the treatment. It should be followed by appropriate irradiation.

Recurrence.—Recurrence is the most disappointing feature. Cheatle and Cutler find that 50 per cent of recurrences take place within the first six months, and that the remainder may appear up to twenty-five years after removal of the initial lesion. The percentage of cases which recur is estimated by Geist and Wilensky to be 37. In addition to a high incidence of recurrence there is also the problem of multiple reappearance to be considered. Gross states that he has operated twenty-two times for fifty-one recurrent mammary sarcomata. Other recurrences are less in number, but two and three per case are far from uncommon.

Of interest is Cheatle and Cutler's observation that the histology of the primary and recurring tumours may be distinctly different. They say "Cellularity and malignancy may increase with recurrence, but not always. Round and polyhedral cell types recur most often, most rapidly, and have the worst prognosis".

Metastases.—As has been pointed out, of the sarcomata in general only lymphosarcoma and melanosarcoma commonly metastasize to the lymphatic nodes. One decimal twenty-eight to 3 per cent of cases metastasize to the axillary lymphatics. There is direct metastasis to distant organs in 12 to 18 per cent of cases. These in order of frequency are the lungs, liver, brain, dura, pleura, mediastinum, retroperitoneal lymphatics, heart, kidney, and bone. Metastasis, as might be anticipated, is most common in the round cell and cellular, large, spindle-cell types (Cheatle and Cutler).

Prognosis.—The prognosis depends upon (1) the extent of the lesion; (2) the complete removal and after-treatment; and (3) the age of the patient (the reverse of carcinoma). Cheatle and Cutler say that, first, sarcoma in a functionally active breast has more recurrence though less metastasis. For sarcomata in breasts of declining activity the reverse is true. Secondly, the prognosis is further influenced by the histological character and stage of development. We have pointed out that this is an element most difficult to establish definitely. Gross says that the average post-operative survival period is eighty-one months; in round cell sarcoma, fifty-four months; in giant cell sarcoma, one hundred and eight months. He gives thirty-seven months as the average expectancy in carcinoma. In general, the prognosis of sarcoma is better than that of carcinoma. Again quoting Gross, the average is 18.8 per cent cure in sarcoma as against 10.4 per cent cure in carcinoma. The latter figure is not above question.

Since 1900, there have been 6 cases of sarcoma of the breast in the wards of the Montreal General Hospital. Four of these have been within

the last twelve years. The other two were discovered in the year 1908, and were described as mixed-cell sarcoma and alveolar sarcoma, respectively. As the slides of these two cases are no longer on file we shall attempt no description of them, but will confine our report to the other four specimens.

Of our four cases for presentation two were males. Two gave history of recurrence. To date there has been no case of sarcoma of the breast in this hospital with metastases.

Our percentage of sarcomata in all new growths of the breast for the past twelve years is 0.6. One per cent of all our malignant neoplasms of the breast for that period are sarmata. We wish to point out that these figures, while lower than those of the earlier writers, are within the general sphere of the later ones.

Our cases are as follows.

CASE 1

M.F. (M.G.H. No. 2537-22, S-22-474) male, aged 70 years; service of Dr. Bazin.

The patient had a swelling in the left breast in the region of the axilla, which had been removed three years previously. A year ago it had recurred to a sufficient size to be noticeable, so it was removed again, and he was told that the pathological diagnosis was "fibroma". Within the year preceding the present admission a second recurrence had appeared in the same position. At operation, a mass, 1.5 by 1.8 by 3 cm. was removed, and diagnosed as spindle-cell sarcoma, with infiltration of the muscle and fat. A radical mammectomy was subsequently performed, and the biopsy diagnosis was reaffirmed.

On microscopic examination, the tumour consists of a moderate cellular tissue, predominantly spindle cells arranged in interlacing strands. These tumour cells belong to the connective-tissue series. On the side nearest the skin the tumour is, on the whole, rather sharply demarcated, from the corium, yet is not encapsulated. On the opposite side there is distinct invasion and replacement of the adjacent muscle. There is no suggestion of encapsulation in the latter area. There are a good many well differentiated blood vessels in the substance. While the tumour cells are well differentiated, and there is no evidence of rapid growth, the invasion of the muscle, coupled with the recurrence, labelled this a malignant neoplasm. *Diagnosis.*—Fibrosarcoma of the breast.

CASE 2

M.McA. (M.G.H. No. 4404-27, S-27-1053 and No. 1767-29 S-29-374). Female, aged 50, unmarried; service of Dr. Bazin.

In 1927 this patient had had a painful lump in the right breast for three weeks. It appeared in the lower outer quadrant, was the "size of a plum", and was unattached to skin, muscle or nipple. There was no discharge from the nipple. The left breast was normal. The lump was removed by retro-mammary approach, and was described as being firmly encapsulated, 1¼ inches in diameter, with small areas of subcapsular hæmorrhage.

"Sections show a moderately cellular tumour, which is sharply demarcated from the surrounding tissue, but does not show a definite capsule. The tumour cells are spindle-shaped, run in interlacing and intersecting bands, are well differentiated, and have the histological character of a slow growth. The adjacent breast tissue shows mazoplasia. *Diagnosis.* — Fibrosarcoma of low-grade malignancy".

In 1929 the patient returned three weeks after the appearance of a second tumour in the same breast, the size of an "olive", firm, nodular, and fixed to the immediately surrounding breast tissue, but not to the skin, nipple or muscle. There was no discharge from the nipple. The axillary glands were not palpable. The specimen was removed, again by a retro-mammary approach, and was found to be 5 x 3 cm. in diameter, white, firm, and apparently composed of fibrous connective tissue.

"Sections show a tumour of the same type as its predecessor, though more cellular. There is now no suggestion of a capsule, and at the margin of the tumour the adjacent tissue is definitely infiltrated. At one point a duct and its acini have retained their morphology, despite their being completely engulfed. The tumour does not have a rich blood supply, though in places there is hæmorrhage into its substance. Moderate round-cell infiltration exists along part of the boundary between the neoplastic supporting tissue. There are many plasma cells throughout this tumour. The adjacent breast tissue shows evidence of mazoplasia. *Diagnosis.*—Fibrosarcoma."

This patient went to another city where she was operated upon in 1932 and another tumour was removed from the same breast, a radical mastectomy being performed. This tumour was diagnosed as carcinoma. We have had the privilege of seeing the sections of this tumour and are convinced that it is of the same type as the tumours described above. The last operation was followed by x-ray treatment.

The woman was seen by Dr. Bazin for the last time about one year ago, on which occasion examination revealed no evidence of recurrence, or metastasis to either the lymphatics or the distant organs.

CASE 3

J.E.F. (M.G.H. No. 727-29 S-29-142, Fig. 1). Male, aged 52; service of Dr. McKim.

The patient had noted a small lump in the left breast for two years prior to admission. It had grown steadily but had produced no pain, and no discomfort. The left breast was larger than the right. The mass did not appear to be attached to skin or underlying tissue; there were no palpable glands in the axilla.

Measuring 14 by 8 by 8 mm., it was well encapsulated and shelled out easily at operation.

"The microscopic section consists of a mixture of collagen fibres and small spindle cells, interlacing, and composing about two-thirds of the tissue. Fat cells among which the spindle cells have infiltrated compose the other one-third (Fig. 1). No epithelial element can be made out in this tumour. No encapsulation is demonstrable, and at the edge of the tumour the surrounding tissue is invaded. The tumour cells are of the spindle-cell type. *Diagnosis.*—Fibrosarcoma."

CASE 4

Mrs. E.C. (M.G.H. No. 7370-32 S-33-22). (Figs. 2, 3, 4.) Female, aged 42, married; five pregnancies; service of Dr. Eberts.

The patient had been previously admitted to the hospital in 1927, *i.e.*, five years prior to this admission. At that time she was suffering from tuberculous periurethritis. In 1928 she was readmitted and nephrectomy and ureterectomy were done for tuberculosis of the right kidney. On the occasion of these admissions she gave a history of no previous serious illness. General examination at these times included the breasts, and note was made of a "congenital marking" above the nipple on the right breast. She could not remember when this had not been there. A small lump was found subjacent to this marking. In June, 1932, she first became aware of the presence of another small lump below the right nipple. At first this grew slowly, but in mid-November it suddenly increased its rate of progress and simultaneously the skin over it took on a bluish colour. She presented herself for admission in December, 1932, because of a "raw" surface which had appeared three

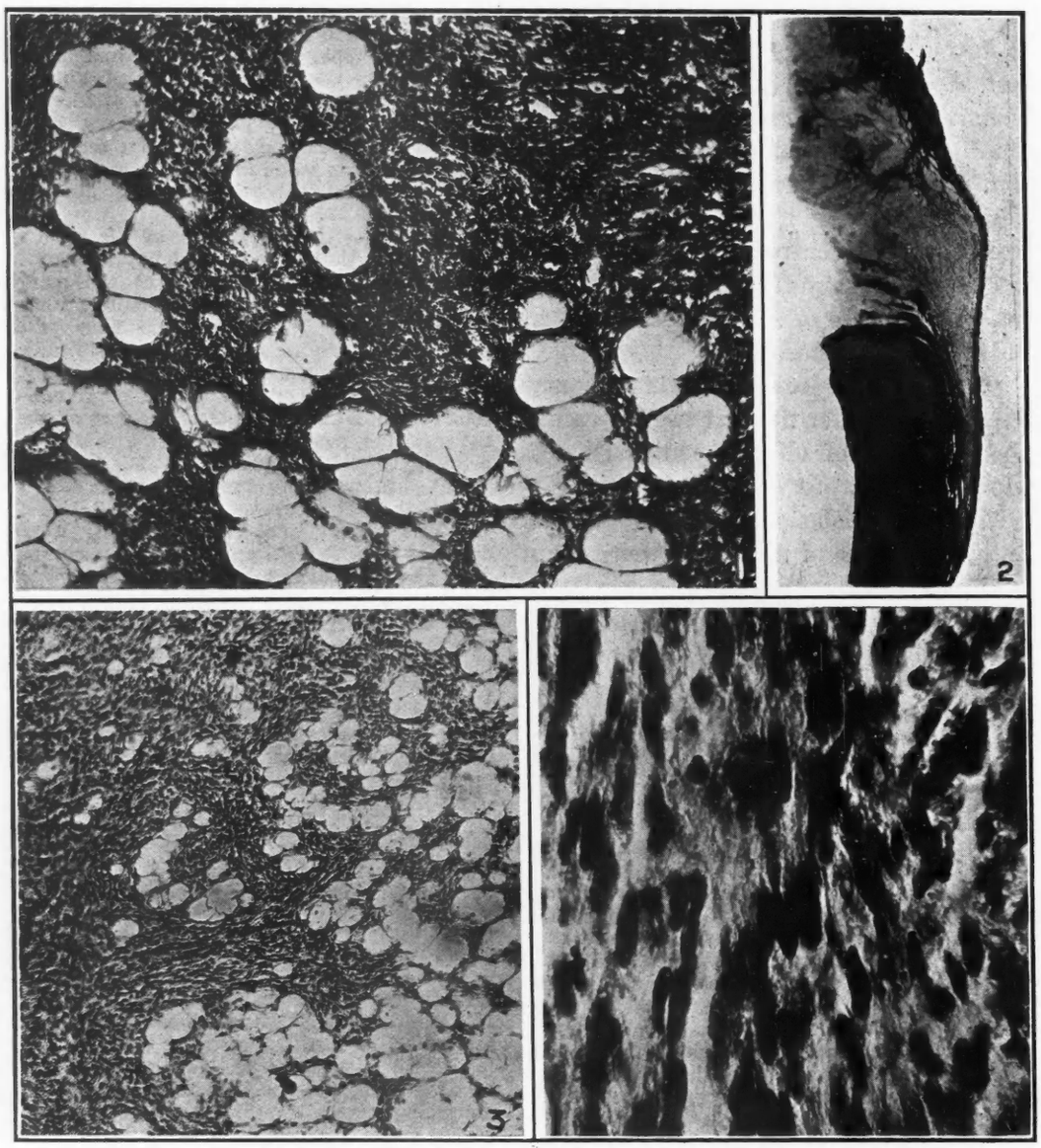

Fig. 1. Case 3 (S-29-142).—Photomicrograph of a tumour of the male breast showing the infiltration of the fat about the gland. **Fig. 2.** Case 4 (S-33-22). — Photograph of large microscopical section of breast showing skin, subcutaneous tissue, and tumour. **Fig. 3.** Case 4 (S-33-22).—Low-power photomicrograph of margin of tumour to show the infiltration of fat. The tumour cells are spindle-shaped. **Fig 4.** Case 4 (S-33-22).—High-power photomicrograph of the tumour cells. Note that these are of the spindle-cell type.

days previously. At no time had there been any pain in the gland or discharge from the nipple.

Examination showed a large pendant deformity of the inferior half of the whole right breast. Beneath an area of bulging granulation tissue an irregular mass, bluish on its surface, and in size 11 by 8 by 6 cm., occupied the most of the organ; it was not attached intimately to the underlying chest wall. Two small nodules were palpable just above the nipple. These were freely moveable. There was one enlarged lymph node in the axillary region and another just above the clavicle on the same side. Examination of the left breast and the remainder of the body was negative for evidence of sarcoma.

Special examination of the blood by Dr. O. C. Gruner showed "Cytomorphological reaction; chromoplasmic changes in 40 per cent of neutrophiles; cytolytic changes, slight; cytase depletion in 6 per cent of neutrophiles; azurophilia in 24 per cent of mononuclears; conditionally specific bodies, plentiful; conclusion, grade 4 malignancy."

A biopsy was taken and a diagnosis of spindle-cell sarcoma was made. A radical removal of the breast was then performed. The remaining tissues were irradiated interstitially with a total of 7,342 mg. hours (radium element). The skin excision was so extensive as to require grafting, the Reverdin type being used. These grafts were successful.

The patient has been seen within the past week, and thorough examination was negative for metastasis or recurrence. She had gained 35 lbs. and felt very well, except for occasional trouble with her one remaining kidney. Of interest is the fact that she has developed keloidal scars, both in the wound and at the point of entry of the radium needles.

"Microscopic examination (Figs. 2, 3 and 4) of the slides reveals an extremely cellular spindle-cell tumour, with strands in some areas, in other places entirely devoid of regular arrangement. The tumour is definitely more cellular near the border where the surrounding fatty tissues is infiltrated. At the margin of the tumour a few fat cells have been incorporated by the advancing growth.

Numerous blood vessels, both large and small, are contained in the neoplasm. Near the centre of the tumour the cells appear to be degenerated. At no point can any epithelial tissue be discovered.''

A section through the ulcerated surface and the adjacent intact skin showed the following. ''The subjacent tissue is infiltrated with polymorphonuclear leukocytes, intermingled with which are numerous eosinophiles. These eosinophiles are especially prominent well beneath the surface of the ulcer. The subepithelial tissue is almost completely replaced by infiltrating tumour cells. The edge of the tumour in other sectors is surrounded by a condensation of the breast stroma which resembles a capsule but which is not. Spindle cells may be seen invading this pseudo-capsule in many places.''

That the ulceration in the skin was due to pressure is very nicely shown in one of our sections. The gradual diminution of the size of the projecting papillæ of the skin as the eroded area is approached clearly indicates that a pressure from beneath is acting upon the epidermis.

A section of the large gland from the axilla showed it to be in an early active stage of tuberculosis, with many small tubercles and Langhan's giant cells. *Diagnosis.*—Fibrosarcoma of breast; tuberculosis of axillary lymph nodes.

COMMENT

The investigation of these four cases has brought out several points of great interest. First was the unexpectedly large number of primary breast sarcomata reported in the literature, together with the high percentage of incidence given by certain authors. Our own figures coincide more nearly with the lower estimates of the more recent studies. We feel that the shift in estimates must be due to more refined differentiative methods. The fact that two of our four cases are males is, of course, coincidental. Second in interest is the fact that in none of our tumours can we demonstrate an epithelial element—either hyperplastic or atrophic. In other words, our tumours in their final form are all ''pure'' sarcomata of the fibrosarcoma type. These are the rarest type of breast sarcomata.

The first three patients were over fifty years of age, one being seventy. The etiology was obscure in all of our cases. Positive diagnosis was made in none prior to examination by the pathologist. In two of our cases recurrence of a previously removed growth indicated the trend of cellular neoplasm being dealt with. Fortunately, there has been no instance of metastasis in any of the cases up to the time of this writing.

The increase in rapidity of growth which accompanied the recurrence in cases 1 and 2, and the distinct change in the type of growth in case 2 are important from a pathological standpoint. These cases also demonstrate that local excision of an apparently benign tumour will not preclude the existence of a potentially malignant cell outside of the capsule. Case 2 also shows that an entire duct system may be completely surrounded by the advancing edge of a sarcoma, yet still retain its identity.

None of our cases shows that element described as being so typical of primary mammary sarcoma, *viz.*, sudden rapid growth in a nodule which had existed previously for some time.

Case 4 demonstrates the rapidity of growth possible in sarcoma, the erosion of the skin by pressure without infiltration, the superabundant supply of thin-walled vessels, which so easily rupture and produce hæmorrhage or are invaded by tumour cells, only to produce distant metastases. This case further shows how neoplastic cells may grow out and completely surround fat cells. It shows the gratifying effect of radical surgery with irradiation on large, ulcerating, malignant tumours in suitable, youthful subjects. The fact that no note is made of a lump beneath the nipple on two previous examinations, together with a definite statement by the patient herself that none was ever there, convinces us that this tumour arose from no antedating benign growth of any duration. We feel that the tuberculosis in this case had no etiological relationship with the tumour under consideration.

SUMMARY

1. Four cases of primary fibrosarcoma of the breast are reported. Two are in females; two are in males.

2. All of these cases are of the ''pure'' type and show no epithelial elements.

3. Local recurrence occurred in two cases, one male and one female. There were no cases with metastases.

4. Our percentage of primary sarcoma of the breast over the period covered (30 years) is 0.6 per cent of all new growths of the breast; 1 per cent of all malignant mammary neoplasms.

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LEIOMYOSARCOMA OF THE ILEUM

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LEIOMYOMATOUS tumours of the small intestine are sufficiently rare to justify the reporting of them. The case cited is the only one to occur in the surgical services of the Hamilton General Hospital during the last fifteen years so far as the author can ascertain. A search of the literature reveals that between 1875 and 1935 not more than 22 cases have been reported, of which 2 occurred in the duodenum, 12 in the jejunum, and 1 in the ileum, the location of the others not being specified. It would appear, therefore, that leiomyoma or leiomyosarcoma of the ileum is a rare disease.

Mr. D., a business man, aged 52 years, was admitted to hospital on June 20, 1935, with symptoms of acute generalized peritonitis. He had been in good health except for a moderate secondary anæmia, the cause of which was undetermined, and which improved greatly under treatment. Two weeks previous to admission he had had an attack of crampy abdominal pain which subsided within twenty-four hours. On June 19th, 1935, while playing golf he was seized with crampy abdominal pain, accompanied by a chill. Unable to finish the game, he went home, and in the ensuing twenty-four hours had several more attacks, each of increasing severity. On June 20th, twenty-four hours after the onset of his illness, an operation was performed by Dr. J. K. McGregor.

At operation, the abdomen showed evidence of generalized peritonitis, and a mass was present which appeared to arise from the wall of the ileum. A portion of the mass was discoloured and covered with a fibrinopurulent exudate. The tumour and four inches of attached ileum were resected, with both ends of the ileum fixed to the abdominal wall.

The first few days after operation were stormy, but the patient showed gradual improvement, and sixteen days later a second stage Mikulicz type of operation was performed in an effort to close the ileostomy. This was partially successful, and was followed thirty days later by a partial anastomosis of the bowel to complete the closure. He was discharged from the hospital completely recovered, nine weeks from the time of admission.

Gross pathology of the tumour.—The specimen consisted of 10 cm. of ileum to which was attached on the serous surface a tumour mass measuring 6 by 5 by 5 cm. One portion of the serosal surface of the tumour was hæmorrhagic and covered with a fibrinous exudate. On section, one portion was fleshy-white and glistening in appearance, while the remainder was granular and showed recent hæmorrhage. The mucosa of the bowel adjacent to the tumour mass showed invasion by the tumour, also a recent perforation.

Microscopic examination.—Sections taken through the greater part of the tumour showed it to be composed of muscle fibres having the arrangement and appearance of that found in leiomyoma of the uterus. In the hæmorrhagic area, the tumour was very cellular, the nuclei rod-shaped, with numerous mitotic figures and very little cytoplasm. The picture in this area was that of a very hyperplastic tumour with marked vascularity. It had the histological characteristics of a leiomyoma with malignant transformation.

Diagnosis.—Leiomyosarcoma.

Fig. 1

Fig. 2

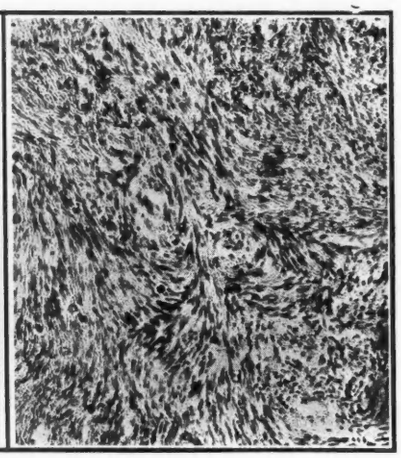

Fig. 3

Fig. 1.—Photograph of the tumour removed showing the external surface. **Fig. 2.**—Photograph of the tumour removed showing the cut surface of the growth and the mucosa of the bowel. The darkened area at the base of the growth is the area of perforation into the mucosa. The darkened area at the upper portion of the growth is the area of perforation and hæmorrhage. **Fig. 3.**—Photomicrograph of section taken from the growth, showing the arrangement of the muscle fibres and the type of cell. The muscle fibres are seen in longitudinal and cross section. The nuclei show variation in size and shape, with occasional mitotic figures.

The patient has recently shown signs of metastases, with marked liver enlargement. Of the 22 cases reported, metastases have been observed in 5, though in a few cases recurrences at the site of the original tumour were noted. It would appear therefore that the great majority of these tumours are either benign or only locally malignant and that complete removal of the primary tumour usually results in cure. They are most common between the ages of thirty and fifty, and occur more frequently in males than in females.

HISTOGENESIS

These tumours arise by neoplasia of the smooth muscle cells which are found in the muscular wall of the intestine, similar to those found in the uterus, blood vessels, and some other organs in the body. The leiomyoma is a common benign tumour of the uterus. Whether true sarcomatous change occurs in these cells is questioned by some, who hold that myosarcomata arise from malignant change in the interstitial fibrous connective tissue, and not in the smooth muscle cells themselves. There does not seem to be adequate support for this view. Cases of sarcomatous change in uterine leiomyomata are on record where the type cell conforms to the leiomyoblastic cell, and these cases show all the usual criteria of malignancy. It is true that active mitosis can be found in the smooth muscle cells of leiomyomata, and that in conjunction with this there is an active hyperplasia of the connective-tissue element which sometimes causes necrosis and disappearance of the neoplastic muscle cells, so that such tumours in the later stages take on the appearance of fibroblastic tumours, but the weight of evidence seems to lead to the conclusion that leiomyosarcomata occur occasionally as malignant transformations of benign leiomyomata.

Andersen and Doob[1] in reporting a case of leiomyosarcoma of the duodenum reviewed the literature on 18 cases previously reported since 1875. In 13 of these, the exact site of origin was mentioned: in 10, the jejunum; in 1, the ileum, and in 2, the duodenum. The incidence was found to be greatest between the thirtieth and fiftieth year. Of the 19 cases, 13 were in men. From reports of the gross structure of these tumours, it appears that they are hard, coarse, nodular, rather non-vascular structures, prone to degeneration in the centre and to ulceration through the mucosal surface of the small intestine. Such ulcerations were described in 7 of the 19 cases. The tumours metastasized in only 4 of the 19 cases, or 21 per cent, and the tumours tend to grow in bulk at their original site rather than to invade adjacent tissues extensively.

Tarry stools, anæmia, palpitation and weakness were the chief symptoms, with vomiting occurring rarely. The x-ray was of little use since the growth of the tumour was external to the lumen of the bowel.

Klopp and Crawford,[2] in reviewing 3 cases, suggest that all tumours of the muscle of the small intestine be called leiomyoma and that, if possible, they be qualified as benign or malignant. Of their 3 cases, 1 recurred after 13 years, and the other 2 were free of symptoms. They suggest that removal of the tumour by operation should give a good prognosis.

SUMMARY

A case of leiomyosarcoma of the ileum is here reported. Twenty-two leiomyomatous tumours of the small intestine have been reported since 1875.

Invasion of the mucosa of the ileum was not advanced, and the absence of tarry stools made clinical diagnosis difficult.

The tumour gives the histological evidences of malignancy, though apparently localized.

Leiomyomatous tumours, benign or malignant, of the small intestine are rarely found in the duodenum or ileum, but are more commonly seen in the jejunum.

The author wishes to thank Drs. W. J. Deadman and H. A. Ansley, of the Department of Pathology, for their kind advice in the preparation of this paper.

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THE INTERNAL FRONTAL HYPEROSTOSIS SYNDROME

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IN the past twelve years there have appeared in the literature isolated studies of what appears to be a new clinical entity characterized by a hyperostosis of the inner table of the frontal bone. Although such changes are said to have been described in the protocol of an autopsy by Morgagni and Santorini[1] published in 1765, the first in recent times to call attention to this condition was Naito[2] in 1924, followed by Casati[3] in 1926 and Greig[4] in 1928. Their studies were of limited importance, in that they were based upon examinations of radiographs or of dried skulls from the museum, with little or no clinical data. In 1928 Stewart[5] reported upon the findings in three personally conducted autopsies. The first report of a living case was made by Morel[6] in 1930. This was followed, in the same year, by the report of another case by Van Bogaert[7] and still another in 1932 by Schiff and Trelles.[8] The first systematic study of a comparatively large series of cases was made by Moore[9] in 1935. This writer reported 72 cases from the hospitals associated with Washington University, and appears to have made a successful attempt to call attention to this condition as a clinical entity.

Moore's 72 cases were found among 415,944 admissions—an incidence of 0.014 per cent. Morel found the condition in 1.4 per cent of the patients in the insane asylum at which his studies were made. With the exception of the cases of Schiff and Trelles and of Moore, all the patients were insane and of an advanced age. Moore's patients varied in age from 14 to 78, averaging 44. All but 2 of his cases (and these he considered doubtful) were females. Of the combined cases of the previous writers 82 per cent were female.

All the patients presented neurological or psychiatric symptoms of varying degrees of severity. Headache was the most frequent symptom, being found in 62 per cent of Moore's cases. Forty-four per cent of the patients were obese. More than 20 per cent had disturbances of vision. Convulsions, diabetes, deafness and thyroid disorders were found in from 5 to 10 per cent.

The diagnostic sign found in all was a hyperostosis of the internal table of the frontal bone. This may be limited to the vertical plates of the frontal, to the orbital plates, or to both. The intimate attachment of the dura to the hyperostotic bone, and the extension of the latter into the cranial cavity was well described by the earlier writers. They did not, apparently, observe an extension of the hyperostosis outwards into the diploë, an occurrence to which Moore draws attention. It is an interesting fact that, while the posterior wall of the frontal sinus may be involved in the hyperostosis, the sinus itself never seems to be invaded. In all cases the process is halted by the inner side of the outer table and never spreads beyond. The newly formed bone is of a sclerotic type, and in thickness varies from a barely perceptible increase to as much as 4.6 cm., as in one of Moore's cases. There were no evidences of bone destruction or decalcification in any of the cases.

From other diseases of the skull the condition can be differentiated radiologically without much difficulty. Its localization to the inner table of the frontal bone is a prominent diagnostic feature. The condition is not easily confused with bone infection, bone tumour, osteitis deformans, leontiasis ossea or any of the bone dystrophies in which loss of calcium is a feature, or with bone changes associated with the blood dyscrasias.

Morel made serial sections of the brain in one of his cases, and found, in the wall of the third ventricle, peculiar cell changes characterized by an increase in pigment and fat. To these changes and to the hyperostosis he attached a common etiological factor. The underlying cause of the hyperostosis is, according to Morel, a generalized decalcification of the bones due to the inactivity associated with old age. The localization of the hyperostosis to the frontal area is, according to Morel and Greig, determined by a

negative pressure produced in this area by traction on the dura, the result of frontal lobe atrophy associated with old age (the average age of his patients was 70), and favoured by the dorsal decubitus. The low blood calcium found in our case, the firm adhesion of the dura to the hyperostotic bone reported in some of the autopsied cases, and the occurrence of this condition in early life cast doubt on this plausible hypothesis. There is a general agreement that the disease represents an abnormality of the calcium metabolism and secondarily of the fat metabolism, as suggested by the frequent occurrence of an obesity which may be extreme. The preponderant, if not exclusive, limitation of this condition to females suggests to Moore the

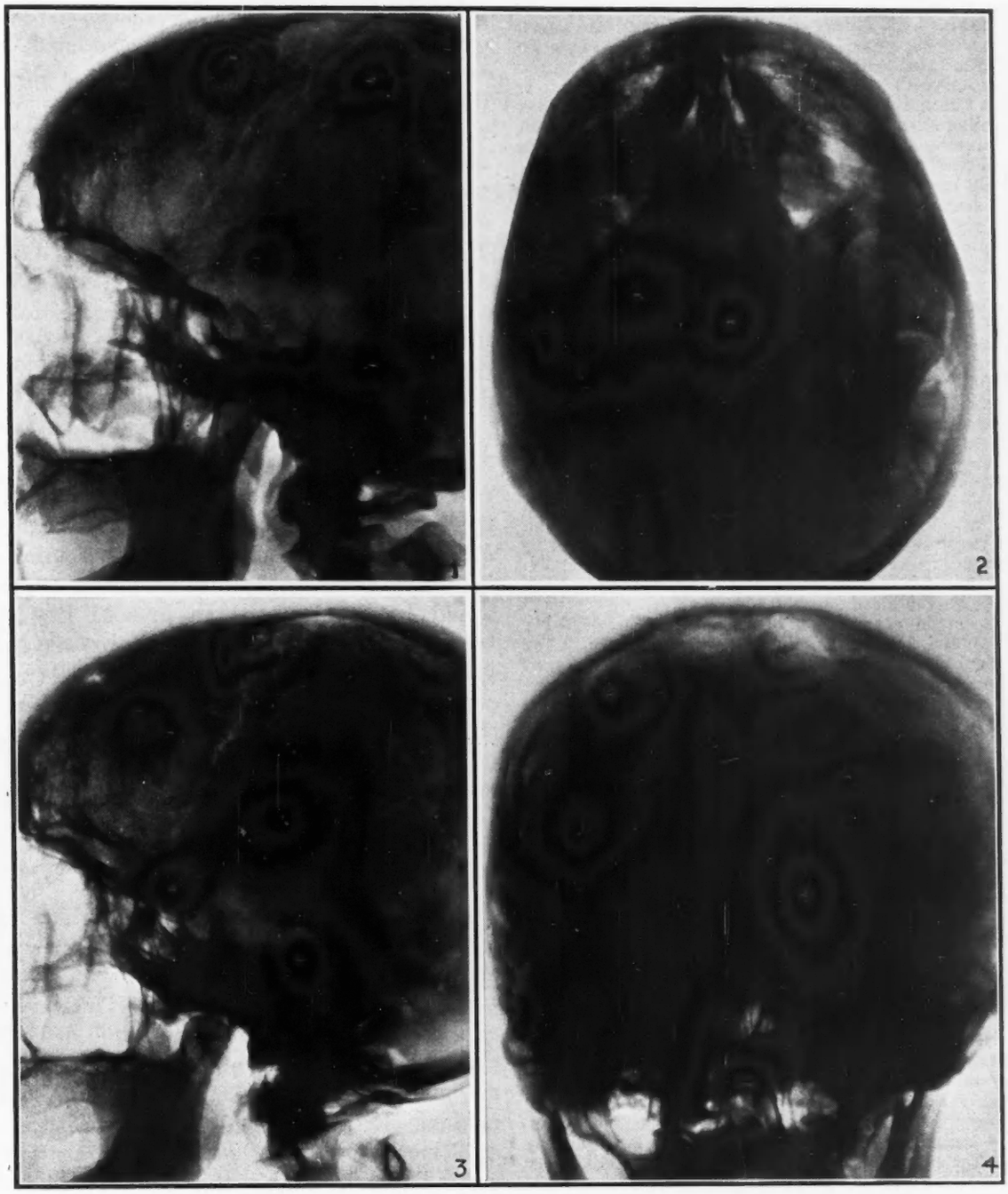

Fig 1.—Film (No. 4625-15: 11. 16. 34) showing hyperostosis of the internal table of the frontal bone. **Fig. 2.**—A tangential view of the frontal bone showing hyperostosis laid down on the inner table of the frontal bone. **Fig. 3** (January 25, 1936).—Showing a slight increase in the thickness of the newly formed bone on the inner table of the frontal. **Fig. 4.**—A postero-anterior view to show the marked thickening of the frontal bone.

possible existence of a controlling mechanism for calcium metabolism peculiar to women, disturbance of which may lead to aberrant deposits.

CASE REPORT

Mrs. D.Z., a married nulliparous woman of 45, was admitted to Mount Sinai Hospital of Toronto, on the Service of Dr. M. A. Pollock, on November 6, 1934. She had been blind in the left eye for two years, but stated that she occasionally saw double. One year before admission she began to lose weight, reducing from 168 to 150 pounds at present. Aside from the above she was in fair health until six months previously, when she began to feel weak. Two months later she began to notice polyuria. Her main symptoms began three weeks before admission with the onset of headache. Shortly afterwards she noticed attacks of dizziness, associated with trembling seizures and mental confusion rather than with true unconsciousness. Her previous history, aside from a gall-bladder operation in 1929, was negative.

On examination the patient was found to be 4 feet 10 inches in height, weighing 150 pounds. Her temperature was 97.0°; pulse 80; respirations 20; and blood-pressure 178/88. The patient showed a tendency to facetiousness, suggesting the "Witzelsucht" associated with frontal lobe tumours. There was a complete deafness in the left ear. Examination of the eyes by Dr. M. K. Bochner revealed slight prominence and slight limitation of movement outward, and poor vision in the left eye. There was no other abnormality and no ocular evidence of increased intracranial tension. There was a diminished sense of smell on the left side. The mouth was edentulous, except for the lower anterior teeth. The throat was negative. The heart and lungs were negative. The vessels were slightly thickened. The abdomen revealed no abnormality. The extremities were normal.

The urine showed a small amount of sugar and a trace of albumin. The blood Wassermann and Kahn tests were negative. A spinal tap showed the fluid to be clear and under normal pressure. The serological examination was negative. The blood non-protein nitrogen was 0.026; the blood sugar, 0.154.

Roentgen examination of the skull (No. 4625-15: Figs. 1 and 2) revealed the following: "The base of the skull shows no abnormality. There is no roentgen evidence of increased intracranial pressure. The outstanding pathological finding is a marked hyperostosis, limited, apparently, to the inner table of the frontal bone. In some place this new bone is about 1 cm. thick. It is not evenly laid down, but, in stereoscopic films, can be seen to form ridges and valleys. This thickening of the frontal bone is well shown in a submento-vertical projection showing the frontal bone tangentially. The rest of the calvarium appears to be negative. The left mastoid is markedly sclerotic and shows practically no cellular formation. All the sinuses are negative. *Conclusion*—The roentgen appearance is that of bone proliferation on the inner table of the frontal bone. The underlying pathology cannot be specifically stated." Roentgen examination of the femurs for evidences of decalcification or other change was negative.

In spite of the lack of evidences of increased intracranial pressure it was felt that exploration was indicated. This was, however, refused by the patient. She left the hospital on December 15, 1934, unimproved. From June to August, 1935, the patient was seen in the Out-patient Clinic. Her complaints were of headache, pain over the right eye, "blindness" in the left, and a severe burning sensation in the right leg from hip to toes. The blood-pressure at this time was 224-110. Her weight, on a diabetic diet, was reduced to 129 pounds.

She was next seen in my office on January 25, 1936. She complained of a return of the convulsive attacks of the previous year. Her symptoms were unchanged. Her weight was 119. A sample of blood for calcium showed 6.3 mg. per 100 c.c. Roentgen examination of the skull (Fig. 3), and a comparison with the films taken 13 months previously revealed a definite increase in the hyperostosis. This was effected by a growth in both directions, *i.e.*, outwards into the diploë and intracranially. There was an average increase of about 2 mm. in thickness, but this was not evenly distributed. A definite thickening of the posterior wall of the frontal sinus and of the orbital plates was now observed. The process seemed to stop at the coronal suture. A postero-anterior film (Fig. 4) showed the process to be apparently limited laterally to the frontal bone.

This case seems in every way to be characteristic of this condition. The comparative frequency of obesity and diabetes associated with an abnormal deposit of calcium in a disease limited almost exclusively to women would suggest a metabolic disease of a somewhat protean nature. The low blood calcium in this case may be of some significance. In the absence of a careful study of a large series of cases any hypothesis as to the pathogenesis of this disease would be merely speculative. The peculiar and characteristic localization of the process to the internal table of the frontal bone is also something about which we can only conjecture. The disease appears to be compatible with long life, but seems to be associated with a progressive mental deterioration.

As far as I know no attempt at treatment of this condition has been made. By analogy, Moore suggests a rachitogenic diet, reinforced by the use of parathormone. This treatment would be based upon the premise that the calcium in the newly-formed, hyperostotic bone was less fixed than that in the normal bone, a supposition that has not as yet been demonstrated. In cases that show definite evidences of increased intracranial pressure, choking of the optic nerves in their foramina, or Jacksonian attacks, Moore recommends operation.

SUMMARY

I report here a case showing the internal frontal hyperostosis syndrome, namely, a characteristic hyperostosis associated with neuropsychiatric changes and obesity. This patient also had diabetes, hypertension and defective vision, defective sense of smell, and deafness, all on the left side. There was a low blood calcium. Radiographs of the skull, taken after a 13 month interval, showed a progressive increase in the thickness of the newly-formed bone. This seems to be the fifth communication reporting a study of a living case, and the first, so far as I know, wherein such a case has been observed for more than a year.

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EFFECT OF REPEATED AND PROLONGED STIMULATION OF THE VAGUS NERVE IN THE DOG*

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IN an earlier paper (1936)[7] we reported that repeated and prolonged intravenous injection of acetylcholine produced in the dog hyaline degeneration of the media of the coronary arteries, with thrombosis and infarcts of the myocardium and evidences of hyaline degeneration and fibrosis. This paper deals with an attempt to reproduce these effects by repeated and prolonged stimulation of the vagus nerve in the dog.

METHOD

Fifteen dogs were used in the course of the experiment. The cervical vago-sympathetic nerve was stimulated electrically as frequently as possible during the survival of the animal. In early experiments the nerve was stimulated for 7 hours continuously, about every fifth day. We later changed to a period of about 2 to 4 hours, 4 to 6 times weekly. Either vagus nerve was used; sometimes one was stimulated for several weeks, and then the other.

Two methods of stimulation were attempted: (*a*) stimulation from a secondary coil buried in the neck, by a modification of the method of Loucks[11] (1933), and (*b*) stimulation by means of shielded electrodes, a modification of Cannon's method[4] (1933), which were fastened to the vagus nerve in the neck and allowed to protrude through a stab-wound in the skin.

A. Secondary coil implantation.—Ten dogs were used; 31 coils were implanted. A coil consisted of two to four thousand turns of 0.004 inch insulated copper wire wound in a coil of an outside diameter of ¾ to 1 inch. The free ends of the coil were soldered to gold-platinum alloy wires of about 0.01 inch gauge, insulated by being dipped in latex (see Fig. 1). These were allowed to project ½ inch to 1 inch beyond the edge of the coil. The latex insulation was stripped off the free ends of these wires for about ½ inch. The bared ends were thrust through a small rubber foot made of a piece of soft rubber tubing 1-1.5 cm. long, slit lengthwise; they were then tied with thread to the inside of this rubber foot, so that they encircled three-quarters of its inside circumference. The whole coil was then insulated by being dipped into celloidin which was hardened and sterilized in 70 per cent alcohol.

The coil was implanted into the neck of the animal, under anæsthesia, by exposing the carotid sheath, separating the vago-sympathetic nerve with as little damage as possible to its sheath, and slipping the rubber foot around the exposed nerve. The slit edges of the foot were sewn together so that the vagus lay in a rubber collar in permanent contact with the bared electrode. The coil was then allowed to lie in a pocket between the neck muscles. The recurrent laryngeal nerve on the same side was cut (so that stimulation of the vagus would not cause spasm of the glottis) and the wound closed.

A few days after implantation the coil and wires became surrounded by a connective-tissue capsule in which a creamy liquid had accumulated. As long as this capsule was undisturbed healing was good, but when the capsule became ruptured the sterile pus tracked to the surface and a chronic sinus developed. The vagus nerve in the rubber collar often became coated with a connective-tissue covering, but usually responded to stimulation so long as the coil was intact. Because of the impossibility of firm fixation of the coil repeated use usually caused a break in one or both wires running from the coil to the rubber foot. Only 7 of 31 coils survived 10 periods of stimulation; of these one was used 19 times and one 22 times. When the implanted coil became defective it was removed and replaced by another on the same or the opposite nerve. Two dogs had 3 coils implanted, two had 4, one had 6 and one had 7.

The vagus nerve was stimulated thus. The animal was given dial or nembutal (because of the acute respiratory distress evident when the vagus was stimulated without anæsthesia), placed on its side, and a primary coil, made by winding insulated 24-gauge copper-wire about an iron core, was held in a clamp close to the secondary coil in the dog's neck. The primary coil was energized by a current of 8 or 11 volts reduced from the 110 A.C. supply in the laboratory by a transformer, the current being made and broken rapidly by an electro-magnetic interrupter. During the period of stimulation the primary circuit was regularly and automatically closed for four minutes and opened for one. This one minute rest was given to avoid fatigue of the peripheral vagus mechanism, and to allow brief recovery from the respiratory disturbance.

B. Shielded electrode stimulation.—Eight dogs were used, 3 of which had previously had coils implanted. The electrodes were made thus. Gold-platinum alloy wire, 0.012 gauge, was dipped in latex. Two pieces about 4 inches long were thrust through a foot made of soft rubber tubing, ½ inch long, 4 mm. inside diameter, slit lengthwise, so that the ends lined the circumference of the rubber foot about 3/16 inch apart (see Fig. 2). These ends were stripped of latex and fastened to the inside of the rubber foot with thread. Since all the

* Read at the Annual Meeting of the American Physiological Society, Washington, D.C., March 26, 1936.

wire was insulated except those parts lying inside the foot, no stimulation of tissues other than that part of the vagus nerve about which the foot was to be wrapped could occur.

The vago-sympathetic nerve was exposed under anæsthesia, the foot sewed around the nerve, the insulated wires drawn through a stab wound in the skin and the incision closed. The ends of the wires were stripped of latex for an inch and connected with the secondary of an induction coil for stimulation. During the period of stimulation the nerve was alternately excited for four minutes and rested for one. In one animal the right vago-sympathetic nerve was stimulated continuously by this method under urethane for 16 hours, 20 minutes, when it died of respiratory failure. The other animals were used repeatedly for shorter periods, as long as they lived.

temper serum was given whenever signs of head-colds or pneumonia developed.

The effects during stimulation.—In order to have a guarantee that the cardiac fibres of the vagus were being excited the nerve was stimulated with an intensity sufficient to reduce the heart rate to one-half to one-third the resting level. Such stimulation provoked sensory disturbances and initiated laryngeal reflexes, thus making it advisable to carry out the experiments in anæsthetized animals. The reactions of the animal during stimulation under anæsthesia varied with the anæsthetic used.

Anæsthetic.—In the early experiments dial was used as the anæsthetic agent. However, nembutal was found to be more satisfactory, chiefly because the recovery period was short and the animals could be

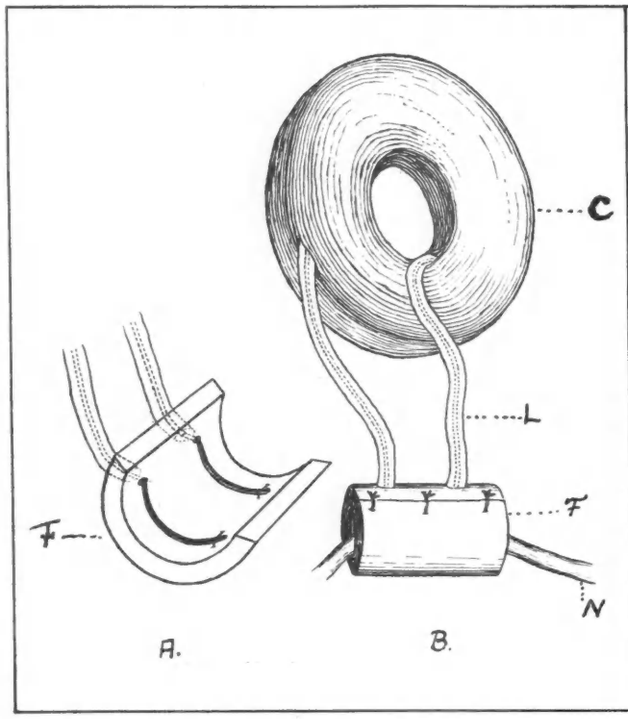

Fig. 1

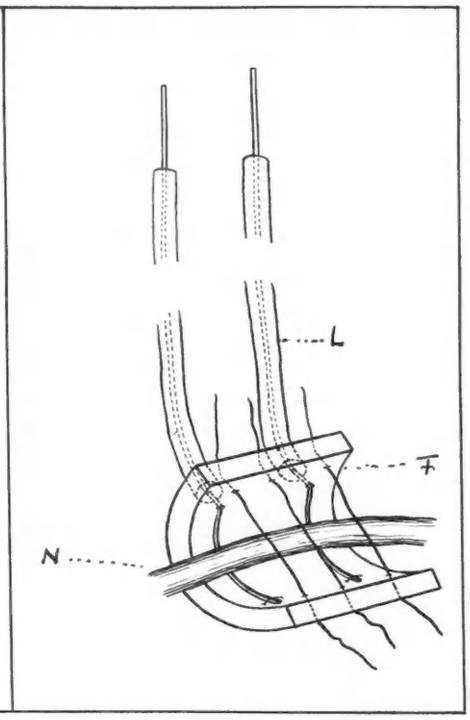

Fig. 2

Fig. 1.—Secondary Coil and Electrodes. The ends of the coil of fine copper wire C. are soldered to the latex-covered leads of gold-platinum wire L, which run to the rubber foot F. Inside the foot the ends are stripped of their latex and fastened with thread to the rubber tubing (A). In (B) the rubber foot F. is sewn around the vago-sympathetic nerve N. **Fig. 2.** — Shielded Electrodes. The latex-insulated gold-platinum wires have been drawn through a rubber foot F, in which they are stripped of their latex and tied to the rubber tubing. They are applied to the nerve as in Fig. 1 (B) and the free ends drawn through a stab-wound in the skin of the neck.

This type of electrode was found more satisfactory than the implanted secondary coil. Ordinarily, the stab wound heals well, without discharge. We found no loss of efficiency of the electrodes in a month of daily use. We also found these electrodes much stronger and less liable to harbour infection than those described by Cannon, which consist of fine silver wire insulated with a piece of fine rubber tubing.

The care and examination of animals.—Preliminary records were made of each animal's weight, arterial blood-pressure (method of Dameshek and Loman,[5] 1932) electrocardiogram, heart-sounds, resting heart-rate, and rectal temperature. After the experiment had started these observations were repeated weekly. The consumption of food and the stools were watched. After a period of stimulation the animal was kept in a warm cage until it had recovered from the anæsthetic. Anti-canine dis-

stimulated more frequently. It was used in all the later experiments. An effective dose gave good anæsthesia for about two hours, and in order to make it possible to continue the daily stimulation for four-hour periods only two injections were necessary. The dogs were usually on their feet soon after the stimulation was stopped, and their appetites were in no way impaired. Intravenous injection did not cause thrombosis. A constant danger, though, was the respiratory depression induced by nembutal; 6 dogs stopped breathing and died shortly after the induction of anæsthesia. Under dial the animal had a higher heart-rate and respiratory rate than under nembutal. Stimulation of the vagus nerve under dial usually accelerated the respiration and slowed the heart; under nembutal both heart and respiration were slowed.

THE EFFECT OF STIMULATION ON THE HEART

The slow heart-rate was taken as an index of vagus excitement. There was usually no difficulty in reducing the heart-rate to one-half or one-third the resting level. Immediately upon stimulation the heart stopped beating, often for 30 seconds, then beat slowly but at an increasing rate which, at the end of 4 minutes was about one-half the normal.

LATE EFFECTS OF STIMULATION

Either the repeated stimulation of the vagus nerve or the repeated and prolonged anæsthesia particularly under dial rendered the dogs liable to respiratory infection, from which nine of the fifteen died. The other most common cause of death was the depressant action of nembutal; some of the dogs ceased breathing shortly after this anæsthetic had been administered, (with the same care with which it had been repeatedly given to the same dog), or after the vagus had been stimulated for a short time when the animal was under its influence. Pneumonia or death under nembutal ended the experiments on every dog prematurely, (except in one animal dying of peritonitis). In six of the animals the experiments were terminated within a month.

The particulars of the stimulation are tabulated in Table I, in which the animals are listed in increasing order of total hours of vagal stimulation. The last ten animals (Table I) might be assumed to show any results of chronic vagus over-excitement.

Three of the dogs exhibited soft blowing systolic murmurs similar to those which we described after repeated administration of acetylcholine. No other clinical signs of impairment of the cardio-vascular system were evidenced, with the exception of the bleeding from the gastro-intestinal tract previously described. The blood-pressure remained normal, and no electrocardiographic changes were produced except in one animal in which bouts of ventricular extrasystoles were recorded. These may have resulted from inadvertent pressure on the carotid sinus at the time the records were taken. The weights of the animals remained stationary or increased, except in those which had respiratory infection.

GASTRO-INTESTINAL DISTURBANCES

Vagus stimulation frequently caused vomiting, especially under dial. After prolonged stimulation the vomitus was frequently coffee-ground in appearance and contained blood. The animal whose vagus was stimulated continuously for 16½ hours vomited blood-containing fluid after 6 hours of stimulation. Vomiting was repeated frequently and was accompanied by the frequent passage of liquid stools containing blood, until death. Some animals continued to vomit and to

TABLE I

REPEATED STIMULATION OF VAGUS—DOG

Dog No.	*Vagus Used*	*No. of Stimulations*	*Average Duration of Stimulation*	*Total Hours of Stimulation*	*Period of Experiment*	*Cause of Death*	*Remarks*
22	L	1	5 hrs.	5 hrs.	1 day	Anæsthetic pneumonia	
29	L	1	5 hrs. 40 min.	5 hrs. 40 min.	9 days	Pneumonia	
28	L	2	5 hrs.	10 hrs. 30 min.	9 days	Pneumonia	
26	R	1	16 hrs. 20 min.	16 hrs. 20 min.	16 hrs. 30 min.	Respiratory failure	Continuous stimulation under urethane until death.
27	L	3	5 hrs. 30 min.	16 hrs. 25 min.	15 days	Respiratory failure	Under dial.
30	L	5	5 hrs.	27 hrs.	26 days	Pneumonia	
32	R	5	5 hrs. 30 min.	27 hrs. 30 min.	36 days	Peritonitis	?Caused by frequent intraperitoneal injection of dial.
31	R, L	6	6 hrs.	34 hrs.	85 days	Pneumonia	
36	R, L	8	2-7 hrs.	37 hrs.	51 days	Nembutal	Died during stimulation.
33	R	7	5-7 hrs.	41 hrs.	42 days	Aspiration pneumonia; gastro-enteritis	
38	R, L	20	3-7 hrs.	101 hrs. 30 min.	45 days	Nembutal and broncho-pneumonia	
42	R, L	35	2-4 hrs.	128 hrs.	52 days	Nembutal	Died shortly after anæsthesia was induced.
39	L	33	3-7 hrs.	135 hrs.	82 days	Nembutal	Died shortly after anæsthesia was induced.
34	R, L	55	2-7 hrs.	269 hrs.	171 days	Nembutal and broncho-pneumonia	Died shortly after anæsthesia was induced.
35	R, L	77	2-7 hrs.	332 hrs.	200 days	Pneumonia	

pass chocolate-coloured, foul-smelling diarrhœal stools, even for two or three days after stimulation was stopped. This gastro-enteritis contributed to the death of some of the animals; two were found dead with an aspiration pneumonia.

AUTOPSY EXAMINATION

Immediately after death a thorough autopsy examination was made. There were no gross changes in the heart or blood-vessels. Pneumonia was common. Intense hyperæmia of the gastric and intestinal mucosa was common, although no erosions nor acute nor chronic ulcers were found. The mucosal blood-vessels were heavily engorged in patches, and the capillaries in the villi were distended, particularly just under the epithelium. Coffee-ground material was found in the stomach of several animals at death. One dog had an area of the fundus from which the mucosa had disappeared, with a rent through the muscularis by which the coffee-ground material had escaped into the belly cavity. There was, however, no evidence of a cellular reaction, and the destructive changes were regarded as post-mortem events.

Routine blocks of the heart were removed for fixation in formalin salt as follows: (*a*) through the anterior coronary artery and left descending branch (including portions of right and left ventricle, and septum); (*b*) flat sections of the right and left ventricle; (*c*) flat sections of right and left papillary muscles; (*d*) flat section through the intraventricular septum; (*e*) cross sections through small blood-vessels at the base of the right and left ventricles.

Sections were stained with hæmatoxylin and eosin and by Millar's[12] (1933) modification of Kull's stain, to show hyaline degeneration. No characteristic degenerative changes in the myocardium or the coronary arteries were detected. Spotty hyalinization of heart-muscle fibres was found occasionally, but not more often than in control animals. In one animal alone there were a few areas of hyalinization of the tunica media of the small coronary arteries, a condition seen particularly well in dogs which had died after repeated intravenous injection of acetylcholine. This dog had, however, received only one period of stimulation, lasting for 5 hours (D-22) whereas other dogs, after vagus stimulation of even as long as 330 hours (D-35) had no demonstrable cardiac degenerative change.

DISCUSSION

Attempts have been made before to produce pathological changes by repeated vagus stimulation. Shaw[14] (1926) described degenerative changes in the aortic arch of the rabbit following this treatment. Robertson and Derbyshire[13] (1935) failed to produce degenerative changes in the coronary vessels of the cat following anastomosis of the phrenic and vagus nerves. Less direct methods involve irritative lesions of the hypothalamus which are regarded as exciting (or, later, paralyzing) autonomic centres (Beattie, Brow and Long,[2] 1930; Beattie[3] 1932; Keller, Hare and D'Amour,[9] 1933; Keller,[8] 1936; Keller and D'Amour,[10] 1936; Watts and Fulton,[16] 1935). These hypothalamic lesions have resulted in bleeding from the stomach and bowel, coffee-ground vomitus, patches of intense mucosal congestion, erosions, acute ulcer formation and perforation. The results are difficult to interpret in terms of irritation or paralysis of specific nerves. Watts and Fulton, for instance, regard the bleeding and erosions which they produced in the gut as evidence of disturbance of the sympathetic vaso-constrictor innervation, although during the operation the heart stopped for as long as 30 seconds "when the lesion was being made in the hypothalamus". This view is supported by the observations of Ungar, Contiades and Grossiord,[15] that prolonged stimulation of the splanchnic nerve in the dog produces hæmorrhage from the digestive tract. Keller (1936) performed hypothalamic operations on dogs after previous bilateral vagotomy *or* abdominal sympathectomy. He found that the damage to the diencephalon produced hæmorrhagic states without ulceration in vagotomized animals, and acute ulceration of the stomach or duodenum without the typical hæmorrhagic states in the sympathectomized animals. The extensive bleeding without ulceration in our experiments was, however, obviously an effect of vagus irritation, and is difficult to reconcile with these observations of Keller, although, on evidence submitted by Greene[6] (1935) we cannot overlook the possible presence of descending cervical sympathetic fibres in the vago-sympathetic trunk in the dog. Even after repeated prolonged vagal stimulation, however, (D-35, D-34), we were not able to demonstrate any evidence of peptic ulcer.

Failure to reproduce the degenerative changes in the heart and coronary arteries which followed acetylcholine injection may be due to one of several causes. The amount of acetylcholine introduced was doubtless very greatly in excess of that which might be produced by vagus stimulation. The acetylcholine was injected daily in measured amounts; vagus stimulation was not as regular, and the amount of vagus-substance produced doubtles varied from time to time. Acetylcholine following injection was distributed by the coronary circulation to all parts of the hearts. The site of production of acetylcholine in the heart is undetermined, although Armstrong[1] (1935) suggests that it is produced only in the ganglia and not at the post-ganglionic endings; in that case it would be hydrolyzed before it could reach the coronary arteries or any large area of the myocardium. Even though it should be produced at the vagal endings in the

coronary arteries, we have no assurance that those artificial stimuli which are apt to cause inhibition of the heart also excite the fibres to the arteries. Green (1935) has shown that stimulation of the cervical vago-sympathetic trunk in the dog may produce either coronary vasoconstriction (vagal effects) or vasodilatation (sympathetic effects); the results are unpredictable. Finally, the acetylcholine experiments were done without anæsthesia, and these experiments were done under dial or nembutal, the influence of which on the vagus fibres to the coronary arteries is unknown.

We are not satisfied, however, that these experiments have proved that prolonged vagus stimulation cannot cause degenerative changes similar to those we produced with acetylcholine. We hope that some method may be devised by which daily excitement of the vagus nerves may be produced over periods of two or three years, in order that the cumulative effect of the repeated production of abnormal amounts of vagus-substance may be studied.

CONCLUSION

Under the conditions of these experiments prolonged electrical stimulation of the cervical vago-sympathetic nerve in the dog, repeated frequently during periods up to 200 days, failed to produce degenerative changes in the heart or its blood-vessels. Congestion of the mucosa of the gastro-intestinal tract, with capillary bleeding, was common, but neither erosions nor ulcers were produced.

We are indebted to Dr. Leslie Jolliffe for the illustrations.

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ORTHOSTATIC ARTERIAL HYPOTENSION. — Defining orthostatic or postural hypotension as a marked fall in the arterial tension when the subject assumes an erect position, the tension being normal in the horizontal decubitus, G. Bickel and M. Demole record a typical case. This condition occurs principally in men over the age of 40, and is rare in women. The cardinal symptoms are a marked fall in the arterial tension, with vertigo, lipothymia, and mental depression. Other less constant symptoms are bradycardia with an exaggerated regularity of the pulse, disorders of perspiration, chronic diarrhœa, loss of sexual powers, and sometimes a slightly lowered metabolism. Periods of remission occur, but the disease usually becomes persistently chronic. Transitory acute forms of only a few days' duration have been noted in various affections, such as influenza and meningitis. Various complex factors enter into the pathogenesis of postural hypotension. These involve the sympathetic system with its cerebral and spinal centres and peripheral terminations, and the endocrine glands; insufficiency of the anterior pituitary lobe and of the suprarenals is emphasized. The treatment of the condition is unsatisfactory. Acute forms disappear spontaneously with the cure of the causal infection. Chronic forms, owing to their frequent encephalic origin, are extremely refractory to treatment. Intravenous injections of ephedrine, ergotamine, pitressin, and calcium chloride have given varying results; the wearing of an abdominal belt or of elastic bandages on the lower limb has given relief in some cases. In cases of endocrine origin pituitary and suprarenal opotherapy has been of great benefit.—*Rev. Méd. de la Suisse Romande*, January 25, 1936, p. 1. Abs. in *Brit. M. J.*

ESTIMATION OF PROTEINS OF BLOOD PLASMA

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THE amounts of albumin, globulin, and fibrinogen in the blood plasma, and the ratio of albumin to globulin, show striking variations in certain diseased conditions, and in some, for example nephritis, may be of importance in diagnosis and control. It is thus desirable to have available a simple and fairly accurate means of estimating these proteins. A number of methods have been devised, and the only excuse for publication of another is that, while retaining adequate accuracy, the actual procedure seems simpler than most of the others. We present here a combined adaptation of the first colorimetric method of Wu[1] and a recent modification due to Andersch and Gibson.[2]

To lend itself most usefully to the needs of clinical chemistry a method should permit reasonable accuracy, should be rapid, and should require no unusual and expensive apparatus. Analytical results should, if possible, be available to the clinician within two or three hours of the taking of a blood sample; time should not be sacrificed to meticulous and unnecessary accuracy. On the other hand, methods designed to give accurate results are less likely to produce misleading error, even when used without great care. Methods involving measurements of minute volumes of fluid (less than 1 c.c.) should be avoided in clinical chemistry.

It seems to be agreed that the plasma proteins of normal blood can be easily and accurately determined by measurement of the refractometric index.[3, 4] Apparently this accuracy does not hold for pathological material,[5] while many clinical laboratories do not possess the expensive instrument needed for these measurements. Earlier procedures involved gravimetric methods; later on, Kjeldahl-nitrogen determinations were made on the separated protein fractions, nephelometry has been used, and now colorimetry can be profitably employed.

Estimation of plasma proteins involves three separate steps: (1) the preparation of the serum or plasma for analysis; (2) the effective separation of the proteins; and (3) their estimation, when separated. (Methods have been devised for the separation and estimation of "euglobulin" and "pseudoglobulin", and even of different albumin fractions, but their clinical utility is and will remain doubtful until the existence of such fractions in the circulating blood is indisputably established.)

Preparation of the plasma or serum.—When the determination of fibrinogen is unnecessary examination of the blood serum is more convenient. If serum is to be used 5 or 6 c.c. of blood should be taken from an arm vein and allowed to clot. After an hour or longer the clot is broken up and centrifuged. One c.c. of serum is needed.

When fibrinogen determinations are required oxalate or fluoride should be used as an anti-coagulant. Such reagents cause lowered values. Their solutions dilute the plasma directly and also indirectly through resulting passage of water from the corpuscles. This dilution can even result in apparently greater protein concentration in the serum than in the plasma which gave rise to it. For clinical purposes the error is insignificant, provided it is kept minimal by avoiding undue excess of anti-coagulant.[4, 6] One drop of 20 per cent potassium oxalate suffices for 5 c.c. of blood; two drops are certainly adequate (and give an oxalate concentration of 0.4 per cent). It seems desirable to use fresh plasma to prevent the changes which occur in fibrinogen on standing.[7]

Separation of albumin and globulin in serum.—It is usually considered that half-saturation with ammonium sulphate is satisfactory.[3] Complete saturation with magnesium sulphate is equally effective,[1, 8] but is not so easily ensured. Saturation with sodium sulphate has been used,[9, 10, 11] but the procedure must be carried out at 37° C. to obtain the requisite degree of saturation, and is slow. Moreover, it has been recently stated that under this condition some albumin is also precipitated.[12]

When ammonium sulphate is used, the globulin precipitate is difficult to centrifuge, so that direct determination of globulin may be inaccurate. McFarlane,[13] using ultracentrifugal measurements, claims that a single precipitation with ammonium sulphate is inefficient, some albumin being frequently precipitated with the globulin. Nevertheless, until some more efficient and rapid precipitant is found half-saturation with ammonium sulphate must still be considered the method of choice in clinical work.

Separation of albumin, globulin, and fibrinogen in plasma.—The above considerations hold also for plasma, once fibrinogen has been removed. This can be accomplished in two ways, (1) by salting-out, and (2) by conversion to insoluble fibrin.

Removal of fibrinogen by salting-out procedures.—Starlinger has dealt with the earlier attempts.[4] The first quantitative method seems to be that of Reye,[14] who laid down precise conditions of dilution of plasma in 0.6 per cent sodium fluoride, and 28 per cent saturation with ammonium sulphate. Andersch and Gibson[2] were the first to associate a salting-out method with colorimetry. They employ oxalated plasma, diluted 1 in 4, and 25 per cent saturation with ammonium sulphate.

Using the colorimetric method to be described, and checking results by determination of fibrinogen as fibrin, we have carried out some experiments which indicate that there is no sufficiently sharp dividing line between fibrinogen and other plasma proteins (including the indefinite "fibrino-globulin" of the literature) to allow accurate determination of fibrinogen by fractional precipitation with ammonium sulphate. Under other like conditions the amount of protein precipitated is increased by increased concentration of plasma and also of anti-coagulant, or, as is to be expected, by dilution of the plasma with 1 per cent sodium chloride instead of with water. Under the conditions specified by Andersch and Gibson it is doubtful if 25 per cent saturation with ammonium sulphate precipitates all the fibrinogen. Saturation of the order 26 to 28 per cent (varying with the precise conditions) seems necessary . Sufficient precision of conditions to permit accuracy is neither feasible nor necessary in clinical work, since an easier method is available.

Removal of fibrinogen as fibrin. — This elegant method was introduced by Cullen and Van Slyke,[15] and has been used by Howe,[9] Wu[1] and others. Sufficient calcium chloride is added to oxalated plasma to remove all oxalate. After a short interval the gel of fibrin can be easily removed. Dilution with 0.8 or 1 per cent sodium chloride is necessary to give a suitable gel. (Obviously dilution with water would precipitate some globulin.) We have slightly modified the original procedure.

Estimation of fibrinogen by difference.—Fibrinogen has frequently been estimated by difference in the refractometric indices or in the nitrogen-content of the serum and plasma. Obviously, unless careful correction is made for volume change produced by the anti-coagulant grave error can arise. In any case, since the amount of fibrin is of a lower order of magnitude than that of total protein, errors of measurement in determining the latter must become considerably enhanced when transferred to the fibrinogen values.

Colorimetric determination of proteins. — In 1912 Folin and Denis[16] published a method for the estimation of tyrosine in proteins, based upon the development of a blue colour through the reaction of tyrosine (or other phenols) with a special phosphotungstic-phosphomolybdic acid reagent. It was shown later that tryptophane and hydroxyproline also give this blue colour.

Wu[1] suggested the use of this reaction to determine plasma proteins, employing standard tyrosine solutions for comparison. It does not matter whether other amino-acid radicals play a part in the colour-production. Wu stated "We have made no experiment to determine what the colour produced by the proteins quantitatively represents. But since this chromogenic value is a constant for any given protein, the intensity of the colour produced under definite conditions can be used as a measure of the amount of the same protein . . . . The tyrosine equivalents of the plasma proteins of different species of animals are probably different, and should be determined when required. To do this it is only necessary to make parallel determinations on the same sample of plasma by the new colorimetric procedure and by the Kjeldahl method."

In Wu's method the colour determination is carried out in dilute alkaline solution. His equivalents for 1 mg. of tyrosine are 16.4 mg. of fibrin, 25.2 mg. of globulin, and 27.5 mg. of albumin. He noted incidentally that the chromogenic value of protein is increased by treatment with sodium hydroxide for a few minutes, or with sodium carbonate on long standing. Wu and Ling[8] substituted sodium hydroxide for sodium carbonate and obtained a 30 per cent enhancement of colour, which developed more rapidly. The factors became for fibrin 11.3, for albumin 15.8, and for globulin 21.4. Andersch and Gibson[2] further increased this colour enhancement by heating the separated protein precipitates with a small volume (0.5 c.c.) of sodium hydroxide for 30 minutes. Under the precise conditions which they specify they find that the protein/tyrosine ratio for all three proteins does not significantly differ from 13.0.

We have used the Wu technique for a number of years in clinical work, and have recently carefully compared it with Andersch and Gibson's modification. The latter has certain advantages in simplicity of operation, but the method for fibrinogen seemed based on an unsound assumption, as we have already pointed out, while their use of minute amounts of plasma and reagent in part of their procedure seems undesirable. Wu used a special phenol reagent. Andersch and Gibson employ Folin's reagent.[1c] Under the conditions of our modification, with a still greater degree of alkalinity, Wu's reagent gives a pure blue colour very easily matched, while the Folin reagent gives a mauve-blue colour with a grey-tint, less easily matched.*

METHOD

Reagents. (1) *Wu phenol reagent.*—Dissolve 100 g. of sodium tungstate, $Na_2WO_4,2H_2O$, and 25 g. of sodium molybdate, $Na_2MoO_4,2H_2O$, in 700 c.c. of water. Add 50 c.c. of 85 per cent phosphoric acid and 100 c.c. of concentrated hydrochloric acid. Boil under a reflux condenser for 8 hours. If the resulting solution be green, restore the correct yellow colour by adding a few drops of bromine and boiling off the excess. The solution can be diluted to a litre,[8] although we usually use it undiluted.

(2) *Tyrosine standard.*—Dissolve 0.200 g. of pure tyrosine in 1 litre of 0.1 *N* hydrochloric acid, to give a 0.02 per cent solution.

In addition the following are required: 1 per cent sodium chloride solution; a saturated solution of ammonium sulphate containing 56 g. of the crystallized salt in 100 c.c.; a saturated solution of sodium carbonate; 20 per cent trichloracetic acid; 10 per cent sodium hydroxide; 2.5 per cent solution of anhydrous calcium chloride.

Procedure when plasma is used.—Use one drop of 20 per cent potassium oxalate for each 5 c.c. of blood. Centrifuge. Use immediately, or, if allowed to stand several hours, filter off and neglect any precipitate which has formed.

Total protein.—Add 9 c.c. of 1 per cent sodium chloride to 1 c.c. of plasma, giving 10 c.c. of *diluted plasma.* Transfer 1 c.c. of this diluted plasma to a 15 c.c. centrifuge tube. Add 4 c.c. of water and 1 c.c. of trichloracetic acid, and shake thoroughly. After 15 minutes centrifuge. Pour off the liquid, draining it off

* Other colorimetric procedures have been suggested, such as, for example, a method based on the biuret reaction.[12] We have had as yet no opportunity of testing their relative accuracy.

as much as possible. Add to the precipitate 1 c.c. of 10 per cent sodium hydroxide and heat on the boiling water-bath for 30 minutes, with a filter funnel in the mouth of the tube to lessen evaporation. Remove, add 7 c.c. of water, 1 c.c. of phenol reagent, and 3 c.c. of saturated solution of sodium carbonate, in the order named; mix by inversion and allow to stand 30 minutes. Centrifuge (neglecting the precipitate) and compare in the colorimeter with the standard set at 10 mm.

Standard (used for all comparisons).—Add to 2 c.c. of 0.02 per cent tyrosine in a centrifuge tube 1 c.c. of 10 per cent sodium hydroxide, 5 c.c. of water, 1 c.c. of phenol reagent, and 3 c.c. of saturated solution of sodium carbonate, in the order named; mix by inversion, and allow to stand for half an hour. Centrifuge, and neglect the precipitate.

Albumin.—To 4 c.c. of the *diluted plasma* add 4 c.c. of saturated solution of ammonium sulphate, giving half-saturation. Mix thoroughly, and allow to stand (corked) in the oven at 37° C. for 20 minutes, to facilitate filtration. Filter. *The filtrate must be clear.* If necessary refilter through the same filter paper until clear. Take 2 c.c. of filtrate (equal to 0.1 c.c. of the original plasma), add 3 c.c. of water and 1 c.c. of trichloracetic acid, and proceed as for total protein.

Fibrinogen (estimated as fibrin).—Take 1 c.c. of *undiluted* plasma in a centrifuge tube, add 10 c.c. of 1 per cent sodium chloride, then 1 c.c. of 2.5 per cent calcium chloride solution. Stir thoroughly, leave the stirring rod in the solution, and allow to stand 20 minutes. A fairly firm clot forms. Whirl the stirring rod. The clot clings to the rod, and by a little manipulation can be worked into a ball and compressed. Transfer to a clean centrifuge tube, add 5 c.c. or more of 1 per cent sodium chloride, knead the fibrin thoroughly with a glass rod, centrifuge if necessary, and drain off the solution. Add 1 c.c. of 10 per cent sodium hydroxide and proceed as for total protein.

Procedure when serum is used.—Take 1 c.c. of serum, add 9 c.c. of 1 per cent sodium chloride, and mix. Estimate total protein (in this case only albumin plus globulin) and albumin as for plasma.

Notes on the above procedures.—(1) After addition of trichloracetic acid the mixing must be thorough. A glass rod can be used, and then washed off with a drop or two of 3.3 per cent trichloracetic acid. (The concentration of trichloracetic acid employed is only just sufficient to ensure complete precipitation of the protein.)

(2) The necessary determinations can be carried out concurrently with a single standard, if timed to permit completion of all comparisons within a few minutes.

(3) The degree of alkalinity employed appears to give a maximum colour development, though this has only been checked roughly. There is definitely less colour when half or twice the amount of sodium hydroxide is used. There is, however, no significant difference in colour development, whether the time of heating with sodium hydroxide be 30 or 40 minutes. The presence of appreciable amounts of ammonium sulphate slightly lessens the degree of colour developed, but the amount carried down in the trichloracetic acid precipitate is without significant effect.

(4) Maximum colour development is complete in 30 minutes, and thereafter there is no significant change up to at least 60 minutes, either with tyrosine or with protein solutions.

(5) In cases of nephrosis or second stage nephritis, when very low albumin values may be expected, double the quantity of filtrate can be taken for estimation and a subsequent correction made in the calculation.

(6) With pure tyrosine solutions the chromogenic value is proportional to tyrosine content between 0.0075 and 0.035 per cent tyrosine (calculated to the original standard solution).

(7) The proportionality between colour development and concentration of protein has been measured over a wide range. It is linear, but in a peculiar fashion, which we cannot yet account for. This is such that, if the standard is set at 10 mm., and the reading of the protein solution is x mm., then the true reading y (were colour development exactly proportional to concentration) is given by

$$y = 1.1\,x - 1.$$

Proof of this relationship will be adduced later.

Calculation for clinical determinations.—Correct all readings as just indicated.

(A) Albumin % = 46.4/(corrected reading).
(B) Fibrin % = 4.28/(corrected reading).
(C) If the amount of albumin is greater than 2.5 per cent
total protein % = 44.2/(corrected reading).
(C′) If the amount of albumin is less than 2.5 per cent
total protein % = 43.2/(corrected reading).

Globulin is determined by difference.

Proof of the accuracy of this method of calculation will now be given, along with a more accurate but more involved method.

EXPERIMENTAL DATA

Chromogenic value of proteins.—Numerous determinations on known amounts (as determined by Kjeldahl measurements) of total protein in sheep plasma (S.P.) and human plasma (H.P.) and on purified albumin (O.A.) and globulin (O.G.) from ox plasma all fit the linear curve given by the equation

$$y = 1.1\,x - 1.$$

A selection of these values is given in Table I, in which the figures in the last column are those which should be found if colour were absolutely proportional to concentration (as determined from Kjeldahl-nitrogen measurements).

TABLE I

Material	*Mean reading* (x)	*Corrected reading* (y)	*True value*
S.P.	4.10	3.51	3.61
O.A.	4.27	3.70	3.71
H.P.	5.35	4.89	4.81
O.G.	6.54	6.19	6.15
H.P.	6.85	6.54	6.40
S.P.	7.30	7.03	7.22
O.A.	7.62	7.38	7.42
H.P.	8.60	8.46	8.53
S.P.	9.10	9.01	9.03
—	(10.00)	(10.00)	(10.00)
S.P.	10.75	10.83	10.82
O.G.	12.05	12.26	12.30
H.P.	12.70	12.97	12.80
O.A.	14.42	14.86	14.84
O.G.	14.63	15.09	15.20
S.P.	17.30	18.03	18.05
O.A.	24.46	25.91	26.00
O.A.	32.03	34.23	34.00

Estimation of the Protein/Tyrosine factor (R).—The factor for *total protein* has been estimated in two ways. (1) The colorimetric result has been compared with the protein value deter-

mined from the difference in total- and non-protein-nitrogen, using the factor 6.25. (2) A similar comparison was made, determining protein-nitrogen on the trichloracetic acid precipitate. In a number of cases both procedures were used, and identical results obtained. The second procedure is easier, and has been chiefly used.

Since 0.0004 gm. of tyrosine is used as standard the factor (R) is given by—

$$\frac{\text{gm. protein in 0.1 c.c. plasma (or serum)}}{0.0004} \times \frac{\text{Reading of unknown}}{10}$$

or

$$\frac{\text{protein \%}}{4} \times \frac{\text{Reading of unknown}}{1}$$

The value for total protein will of course vary, even for normal bloods, through variations in the proportions of the different proteins present. The following results have been obtained. Duplicate determinations were made throughout, except for human blood.

Ox.—Plasma proteins. 5 samples. Extremes 11.0 – 11.3. Mean 11.1.
Serum proteins. 2 samples. 10.9, 11.2.
Man.—Plasma proteins. 6 samples. Extremes 10.8 – 11.3. Mean 11.05.
Sheep.—Plasma proteins. 1 sample. 11.0.
Serum proteins. 1 sample. 10.9.

For *albumin* solutions were obtained by the Howe procedure—saturation with sodium sulphate at 37° C. and filtration—and protein determined in the filtrate colorimetrically and by Kjeldahl. It was found that the presence of sodium sulphate in the requisite concentration significantly diminished colour development and a curve of correction had to be constructed and applied. The following figures were obtained (from duplicate determinations, except for human blood):

Ox.—Albumin from plasma. 3 samples. Extremes 11.2 – 11.9.
Albumin from serum. 6 samples. Extremes 11.2 – 11.8.
Mean of the 9 determinations 11.5.
Man.—Albumin from plasma. 6 samples. Extremes 11.4 – 12.0. Mean 11.6.
Sheep.—Albumin from plasma. 1 sample. 11.3.
Albumin from serum. 1 sample. 11.1.
Mean of the 2 determinations 11.2.

Determinations for ox blood *globulin* were made on samples of globulin precipitated from serum by saturation with sodium chloride and then brought into solution in dilute sodium chloride. Nitrogen and colour values were compared as usual. The values for three samples were 9.9, 10.0, and 10.2, giving the mean 10.0.

Ox blood *fibrin* was precipitated by the procedure already outlined, and brought into solution in 0.1 *N.* sodium hydroxide. Different portions of the same solution were used for nitrogen and colour determinations, using the factor 5.91 to convert nitrogen to protein.[17] Three samples gave the values 10.5, 10.7, and 10.9, giving the mean 10.7. On account of the large volume of blood necessary for these determinations it was not feasible to use human blood, and the figure for ox blood has been considered sufficiently accurate (*cf.* 18). Commercial fibrin gave the value 11.5, and pure dried preparations of ox albumin and globulin also gave values significantly different from those determined on undenatured material.

To obtain values for human blood globulin, plasma was analyzed by the routine colorimetric procedure, and in addition a sample of the same plasma was analyzed for total nitrogen, and a second used to prepare a pure albumin solution by sodium sulphate precipitation (from this the factor for albumin was determined). The following example illustrates the procedure, which can be followed more easily after reference to the next section. The "inverted" values are

(A) Total protein: Corrected reading 6.15 mm. Inverted reading 1.626
Albumin: " " 10.3 mm. " " 0.971 }
Fibrin: " " 10.5 mm. " " 0.095 } 1.066
Difference, due to globulin 0.560

(B) Total protein: Determined by Kjeldahl 7.23%
Albumin: Factor found 11.6. Percentage (4 x 11.6) /10.3 = 4.50% }
Fibrin: Factor assumed 10.7. " (4 x 10.7) /10.5 = 0.41% } 4.91%
Difference, due to globulin 2.32%

$$\text{Globulin factor} = \frac{\text{globulin \%}}{4} \times \frac{\text{Reading for globulin}}{1}$$

$$= \frac{2.32}{4} \times \frac{10}{0.560} = 10.4$$

obtained by dividing the corrected colorimetric readings into 10, and are of course directly proportional to the colour production.

Obviously this indirect procedure enhances the error of a single determination. With the six samples of blood used the values 9.8, 9.8, 10.0, 10.2, 10.6 and 10.7 were obtained, giving the mean value 10.2.

The above determinations are summarized in Table II.

TABLE II.
FACTORS FOR PLASMA PROTEINS

	Man	*Ox*	*Sheep*
Total protein	11.05	11.1	11.0
Albumin	11.6	11.5	11.2
Globulin	10.2	10.0	—
Fibrin	—	10.7	—

The number of determinations is insufficient to stress any species differences, and it is quite possible that a larger number would show identical values for human and ox blood. In the meantime it seems justifiable to assume that use of the figures 11.6, 10.2, and 10.7 for human albumin, globulin, and fibrin respectively will not lead to significant error.

Calculation of results.—Correct all readings by the equation $y = (1.1\ x - 1)$, as already explained.

For albumin, since the standard, containing 0.0004 mg. of tyrosine, is contrasted against the albumin in 0.1 c.c. of plasma, the amount in 100 c.c. is given by—

$$0.0004 \times \frac{10}{y} \times 11.6 \times \frac{100}{0.1} = \frac{46.4}{y}.$$

A similar calculation can be used for total protein, when its factor is known or can be assumed.

Since the fibrin is determined in 1 c.c. of plasma the calculation becomes—

$$0.0004 \times \frac{10}{y} \times 10.7 \times \frac{100}{1} = \frac{4.28}{y}.$$

The figure for globulin can be obtained in two ways, the first accurate, and the second a rule of thumb procedure which is sufficiently accurate for almost all clinical purposes.

1. Divide the corrected readings into 10, giving "inverted" readings which are directly proportional to the developed colours. Subtract the sum of the albumin and fibrin values from that for total protein. The difference corresponds to the globulin colour, and, being an "inverted" figure, gives the globulin percentage when multiplied by $(4 \times 10.2)\ /10$ or 4.08.

2. If the albumin percentage is greater than 2.5, take 11.05 as the factor for total protein, and divide the corrected reading into (11.05×4) or 44.2. If the albumin percentage is less than 2.5, take 10.8 as factor for total protein, and divide the corrected reading into (10.8×4) or 43.2.

These methods of calculation can be explained most easily by an actual example.

Case L.B. Normal. First Method.

Albumin:	(i)	Reading 12.8 ,	corrected 13.08.	46.4/13.08	= 3.55%
	(ii)	" 13.0 ,	" 13.3 .	46.4/13.3	= 3.49% Mean 3.53%
Fibrin:	(i)	" 14.7 ,	" 15.2 .	4.28/15.2	= 0.28%
	(ii)	" 14.7 ,	" 15.2 .	4.28/15.2	= 0.28% Mean 0.28%
Total:	(i)	" 7.3 ,	" 7.03.		
	(ii)	" 7.65,	" 7.42.		

Albumin:	Mean corrected reading	13.19.	Inverted	0.758	
Fibrin:	" " "	15.2 .	"	0.066	..... 0.824
Total:	" " "	7.23.	"	.....	1.383

Difference (globulin) 0.559 x 4.08
= 2.28% globulin, and 6.08% total protein.

Second method: Since albumin is greater than 2.5 per cent, total protein is given by 44.2/7.23, equal to 6.1 per cent. Subtracting the sum of albumin and fibrin (3.8 per cent) the figure 2.3 per cent is obtained for globulin.

Results obtained by the method.—Some of these are shown in Table III. The figures given in brackets were obtained by the second, rule of thumb, procedure. Only in the last, an unusual case, is there any significant difference. The duplicate figures given for a number of cases indicate a reasonable degree of accuracy for the method. There has seemed to be no advantage in attempting to check these figures by micro-Kjeldahl determinations, since the whole method is based on more accurate macro-Kjeldahl measurements.

TABLE III

SOME RESULTS OBTAINED BY THE NEW METHOD

Subject	Condition	Albumin	Globulin (i)	Globulin (ii)	Fibrin	Total (i)	Total (ii)
		Percentage	Percentage	Percentage	Percentage	Percentage	Percentage
L.B.	Normal	3.55 3.49	2.28	(2.3)	0.28 0.28	6.08	(6.1)
Mixed – Plasma		4.35 4.35	2.12	(2.1)	0.21 0.24	6.70	(6.7)
J.B.	(Prolapse of uterus)	3.74 3.72	2.34	(2.4)	0.31 0.32	6.39	(6.4)
D.Y.	(Indigestion)	3.49 3.52	1.42	(1.4)	0.23	5.16	(5.1)
S.J.	Hypothyroidism	3.71 3.13	3.11	(3.2)	0.21	6.74	(6.8)
P.C.	(Arthritis; bronchitis)	3.15 3.13	2.21	(2.3)	0.28 0.28	5.63	(5.7)
D.F.	Second stage of glomerulonephritis	2.24 2.22	2.23	(2.3)	0.45	4.91	(5.0)
D.R.	Marked post-operative œdema	1.38	3.30	(3.4)	0.27 0.30	4.97	(5.1)
	After treatment; œdema lessened	1.90 2.21	3.30	(3.4)	0.39 0.40	5.74	(5.8)
W.W.	Nephrosis	0.93 0.92	1.38	(1.4)	0.64 0.70	2.97	(3.0)
	After treatment	1.66	1.91	(1.9)	0.53	4.10	(4.1)
R.C.	Presumed normal	4.49	1.17	(0.8)	0.25	5.91	(5.6)

SUMMARY

A simple accurate method is outlined, designed for clinical use in the estimation of albumin, globulin, and fibrin of blood plasma.

It is based upon comparison of the colour developed by these proteins and by tyrosine with Wu's phenol reagent.

Chromogenic variation with concentration has been measured, and a simple correction for this variation has been determined.

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NOTES AND OBSERVATIONS ON HYPERPIESIA*

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THE normal blood-pressure is as variable as all other vital mechanisms in health — a measure for quick bodily adaptation to change or emergency in environment. Persistently heightened pressure is present in a number of disorders; in some renal diseases, in chronic lead poisoning, in hyperthyroidism, in adrenal tumour, in some intracranial diseases, and in other conditions of rarer occurrence. But the most common type of all is that in which no extra-circulatory cause can be found. This has been given many names, of which two seem justly popular—"hyperpiesia", the name originated by Sir Clifford Allbutt; and "essential hypertension", the expression of Sir Thomas Lewis. It is this type alone which is discussed here.

Mosenthal defines hyperpiesia as a disease of unknown etiology, characterized by a persistent and progressive rise of both the systolic and diastolic blood pressure. Allbutt's definition agrees with this, except that progressive rise is not stipulated. Fishberg defines it as including those cases of chronic hypertension which neither clinically nor anatomically can be demonstrated to have evolved from antecedent inflammatory disease of the kidneys or from urinary obstruction. According to O'Hare, the basic fault producing the syndrome is an arteriolar instability of function. Over this vasomotor phenomenon the veil is drawn — what may be the defect, what the aberrant metabolism or control that causes the instability, is unknown. The arteriolar fault begins to show itself in adolescence by easy flushing, coldness and blueness of the hands, and emotional excitability; and in adult life, as restless energy, cloaking a still present instability. One can envision a vicious train—a too-ready and too-enthusiastic pressor response to calls received over-often from the primitive defense mechanism as the fight of living presses on; the too-frequently contracted arterioles increasing peripheral pressure, and thereby pressure in the arteries upstream; arteriolar walls becoming hypertrophic and sclerotic in compensation, and this reacting to make constant high arterial pressure necessary to maintain efficient arteriolar service to the capillary bed. Definitely, however, arteriolar sclerosis has been found a result and not a cause of hypertension.

Experimentally, there are flaws in such an explanation, but, at any rate, the predisposed subject in his "thirties" shows a tendency to easily induced hypertension, and in his "forties", to hypertension without return to normal when relieved from stress. His arterioles now are beginning to show sclerotic changes and are narrowing, with two important results—first, an impaired blood supply to the capillaries beyond, and, second, an increased load on the arteries and the heart behind them. The impaired capillary blood supply leads to ischæmic changes in the tissues concerned, and these to atrophic and sclerotic degenerations. As in arteriosclerosis, the body is not evenly affected. In one patient the ischæmia may be more severe in the kidneys, in another in the heart muscle, in a third in the brain; and the progress of the malady and its termination differ accordingly. Some fortunate patients have their major degenerations in organs less vital than those named, and can outlive their less lucky fellows.

In search of etiology, Sir Clifford Allbutt gave some classic examples of the hereditary tendency to hyperpiesia, and all other writers are in accord with him that the strongest causal factor is heredity, both racial and familial.

The blood-pressure of Chinese has been found to average 20 to 30 mm. lower than that of Europeans and Americans, and the average systolic pressure of Hindus is 100 mm. Dennison, in examining 1,000 natives of Kenya in Africa, found no hypertensives. Whites living in these countries retain their higher pressure averages. But the Hindoo and the Kenyan live no longer than the white man; they merely die differently.

O'Hare, Walker and Vickers, in 1924, elicited a family history of vascular diseases in 68 per cent of 300 patients with hyperpiesia, but in only about half that percentage in normal controls. Wietz found that among 82 patients with hyperpiesia 77 per cent had

* Revised from a paper read before the Saint John Medical Society, February 26, 1935.

lost one or both parents from the consequences of hyperpiesia, and cites an interesting example of female twins who at sixty-three years of age showed an equal hypertension, yet one had led a hard life working in a factory and the other had lived a sedentary existence. Most writers, however, agree that overwork of the nervous, insatiable sort, and long-sustained mental tension, are potent causative factors.

A world of work has been done on the causal relation to hyperpiesia of anatomical peculiarities, endocrine influences, bacterial and chemical poisons and nervous control. Widely diversified pathological conditions have caused it—coarctation of the aorta and arterial hypoplasia (in the sector of back-pressure), adrenal tumour, basophilic adenoma of the pituitary, hyperthyroidism (affecting systolic pressure), and diabetes; but the vast majority of hypertensives have none of these lesions. The sex glands have long been credited with a definite relation to hyperpiesia because of its recognized concurrence with the menopausal period, but Lehfeldt in 1926 noted that, while marked fluctuations of pressure were characteristic of the menopause, only 16 out of 111 women in the series studied were found to develop hyperpiesia, and of these several might have been due to extraneous influences. He concluded that only those already predisposed developed hypertension at this time. The appearance of hyperpiesia in men of the same age has led to no corresponding thesis. In our present state of incomplete knowledge of endocrine activity and of its glandular interactions, and of the operations carried out through its adjutant, the autonomous nervous system, it is impossible to tell definitely how disturbance of internal secretions produces changes in arteriolar tonus, and so hyperpiesia.

Studies in food metabolism have at least refuted the causal theories of cultists. The Eskimos living on meat alone show no more hypertension than the vegetarian enthusiasts. Steffanson lived by periods up to nine months at a time, for nine years, on meat alone, and kept a blood-pressure of 115/55 mm.; a patient of Faber's who had been a strict vegetarian for twelve years had a systolic blood-pressure of 220 mm. Similarly, fats and lipoids and carbohydrates have been tried and found wanting as causative agents, and the relation of salt, potassium, sodium and calcium is still in abeyance.

In regard to extrinsic causes there seems little doubt that chronic poisoning by lead will produce hyperpiesia, through a general vasoconstrictor effect, but other poisons, including nicotine and alcohol, have been found innocent on this charge, at least. Focal infections, including intestinal absorption, have also been found inconsequent. So may the search for etiology be sketched—it almost reminds one of Leacock's remark on the advance of knowledge about appendicitis— "We used to think that grape seeds caused appendicitis; now we know that they don't."

Even the criterion of normal blood-pressure is not yet fully settled. In young adults a systolic pressure of over 130 mm. is considered by Alvarez to indicate a hypertensive diathesis, and 140 mm. is considered the upper limit of normal. Janeway regarded 150 mm. as the normal limit at all ages, and Lewis suggests 160 mm. in older subjects. The systolic pressure is found rather higher than this in many elderly people without an abnormal diastolic rise. This is written of as an effect of rigid arteries, and the judgment as to whether it is to be called abnormally high depends on whether or not arteriosclerosis in the aged is an abnormality. The question is begged as to whether old age is an abnormality. In the sense that such pressure necessarily shortens life beyond the span of three score and ten years it can hardly be considered abnormal, as witness Bowes' figures of pressure readings in 115 aged people. From ages 75 to 79 years there were 14 men with an average systolic pressure of 159 mm., 24 women at 170 mm. average: from 80 to 84 years there were 11 men at 163 mm. and 16 women at 183 mm. average, and 14 more subjects ranging up to 94 years of age—a total of 79 very old people living with high systolic pressures. In all, however, the diastolic pressures were normal. The upper limit of diastolic pressure is placed at 90 to 95 mm., the former figure being given me recently by a Medical Insurance Institute as the latest preference. Palmer names 100 mm. as the limit of normal.

The age of onset is a detail on which one can get very little information. Janeway's statistics showed that 80 to 90 per cent of his cases occurred between the fortieth and sixty-ninth years. This does not make clear the age of onset. Lewis speaks of it as being rare before the age of forty and having its chief incidence in the fifty to seventy-year period. Fishberg considers that the vast majority of cases are established at the age of forty. The Medical Director of a large Life Insurance Company wrote me that he judged that onset occurred

chiefly in the 40 to 50 years' period. The Director of a Medical Insurance Institute kindly tabulated for me a series of 166 cases according to age at which hyperpiesia was first noted. It reads as follows:—

41 to 45 years	48 cases
46 to 50 years	53 cases
51 to 55 years	39 cases
56 to 60 years	12 cases
61 to 65 years	7 cases
66 to 70 years	7 cases
	166

Symptomatology is elusive also, which probably explains why onset is so seldom observed. The common symptoms named by observers are headaches, paræsthesias, especially of the scalp, irritability, dizziness, and epistaxis. These are of established but simple hyperpiesia; later, of course, symptoms of cardiac, renal or cerebral consequences are specialized. Other observers consider that there are no symptoms of simple hyperpiesia, and that those mentioned above are due to concurrent arteriosclerosis. Some authors state that signs of vasomotor and emotional instability — epistaxis, irritability, nervousness and cyanosed hands—seen in the second or third decade, are precursors of hypertension. They find them present in the history of 50 per cent of hypertensives, and in only 23 per cent of controls.

Of signs there are two; persistently raised systolic and, optionally, diastolic pressures; and persistently contracted arterioles. It is very necessary to stipulate persistence in high pressure, for in a series of readings on a case one finds great variations, and I myself have come to disregard a high reading on the first visit to my office, especially if it is accompanied by a pulse of about 120 and other signs of nervousness.

The spasmodic or contracted arterioles are only to be seen in the retina, which, *faute de mieux,* must be used as an index to the general arteriolar system.

It is generally stated that the blood pressures of the hypertensive become progressively higher. In the little series of cases which I have reviewed for this paper I cannot concur in this. In the five years or less of observation there seemed no heightening, and where figures were obtainable of bygone readings, no progressive increase had occurred.

From the literature and from one's own observations it would seem that the great majority of hypertensives, no matter how aged, have had that hypertension since some time before the age of fifty-five, and that their mean pressures have not increased since then; also that the class of cases whose diastolic pressure is raised abnormally has established hyperpiesia a decade earlier than the class of normal diastolic pressure.

As years pass, the hypertensive takes one of several paths, determined by where his arteriolar sclerosis predominates—in his kidneys, in his heart or in his central nervous system; or by whether it is too evenly distributed to wreck any one vital system first. In all paths there is a common effect—the hastening of arterial sclerosis. It is probably this which contributes the first symptoms that lead the patient to our offices—the dizziness, fatigue, dyspnœa, paræsthesias of the scalp and of the extremities, and the nasal and conjunctival hæmorrhages. A healthy arteriole is hard to burst, even with high pressure behind it; hæmorrhage tells of sclerosis in addition. It must not be inferred that arterial sclerosis is caused by hypertension; it is simply hastened. Eighty-eight per cent of cases of marked arteriosclerosis have never had hyperpiesia (Sawada). Unexplainably, the symptoms of this stage are transitory and recurrent—the flatulence is troublesome for a few weeks at a time, the dizziness or headache may be annoying for a few hours, at intervals of months. If they are to be explained as ischæmias from permanent arteriolar changes one would expect them to be more enduring.

Only 10 per cent of deaths are renal, according to Mosenthal. In my own little series the percentage has been much lower, about 2. At any rate, the renal is by far the least common direction for hyperpiesia to take. And it is the earliest, terminating usually before the age of fifty-five. The lesions produced are those of the arteriolo-sclerotic kidney — minute patches of ischæmia going on to equally small atrophic foci comprised of shrunken glomeruli and sclerosed tubules. Fishberg describes a second type of malignant hypertension, in which renal failure may appear later, after a decade or more of established hyperpiesia without renal involvement.

Cases of the renal type may be very difficult to distinguish from chronic glomerulo-nephritis, especially since they occur at the border-time in age between nephritis and "malignant hypertension". The urine in both diseases may be loaded with albumin and casts, including Addis casts; the blood pressure is usually not so high in chronic nephritis, and neither is it so labile; the retinal arterioles do not show as much sclerosis in chronic nephritis; but all these criteria are indefinite. Only a history of previous acute glomerulo-nephritis may help one to differentiate. Fortunately, such renal disasters are comparatively rare, and the other 90 or more per cent of patients travel down the quieter paths in which the kidneys, though nearly always affected, degenerate so slowly that they outlast other organs, and even in old age give a good Volhard test and show trifling urinary albumin and casts.

Sixty per cent take the cardiac way. The heart has to contend against three handicaps; one, the extra mechanical load of pumping against a fluid pressure increased often to double the normal; second, the deleterious effect on its own strength of a gradually increasing myocardial ischæmia; and third, the failure to get its customary diastolic rest because of increased diastolic blood pressure. A slow but unrelenting race goes on between the twin plagues, overwork and undernourishment. From overwork the left heart dilates, and, as it does, the ventricular muscles thicken to meet the strain, and dilatation and the mitigating hypertrophy go on to a time when fatigue of the myocardium can no longer be postponed and congestive failure or a series of failures, patched up by rest, closes the picture.

From undernourishment comes the diffuse fibrosis that degenerates the myocardium and leads to interruption of its conduction system and to arrythmias and blocks, all furthering the loss of power. The concomitant sclerosis of coronary arteries prevents them from averting acute muscular ischæmias when the heart is called on for extra output, and the patient suffers angina; worse still, it may permit the formation of a coronary thrombus and an ischæmic catastrophe, announced by still more severe angina—an infarct of the ventricular wall; a gross necrosis which may kill almost instantly, or may heal and leave a patch of weakened, fibrous, inert tissue in cardiac muscle, already in sore need of all the useful units it can muster.

It is really extraordinary what overwork many hearts stand in hyperpiesia if the ischæmic factor does not overcast the picture. One sees cases of over ten years' recorded duration in which there is yet little hypertrophy, and others in the late "seventies" and over, where the patients probably have been hypertensive for thirty years, whose hearts are neither more than moderately enlarged nor show signs of gross decompensation. The amount of diastolic over-pressure, although mathematically it does not add a great percentage of work to the heart, seems definitely to be the major factor in the heart's power to last. The predominantly overworked heart is the one which may last into old age, and accounts for 44 percent of the total deaths from hyperpiesia—much the largest group. The predominantly ischæmic heart, with sclerosis of the coronary vessels, often does not permit such hypertrophic regrowth of muscle, and fails earlier, usually in the "fifties" and "sixties", the later stages being anginal. The sudden catastrophies of coronary embolism and thrombosis usually develop even earlier, and are to be blamed on arteriosclerosis rather than hypertension, since frequently they occur in the absence of the latter. Fortunately this ischæmic predominance accounts for only 16 per cent of the total deaths.

The third main path into which the hypertensive may drift is the cerebral, and 20 per cent follow this to termination in the "sixties" and "seventies", rarely earlier. Strange things happen to them on the way, but none until arteriosclerosis has fallen into step with the hypertension. Headaches and dizziness are early troubles, and "weights" or "hot spots" on the vertex, and occasional stabs of pain. Word-memory may suddenly fail, or there may be amnesias, or loss of part or all of the visual fields, or a member may become weak or paralyzed or numb. All these may leave after a few minutes or a few days. Good authorities explain them on the basis of spasm of sclerotic cerebral blood-vessels; one of the most exact of the great authorities, Allbutt, claims that they are embolic. They tend to recur, maybe after years. Small blood-vessels, their walls deterio-

rated by atheroma and bulged by internal pressure, suddenly give way in retina or labyrinth and cause blindness that may at best clear up after months, or the terrific vertigo and roaring and vomiting of Ménière's syndrome. Some day a larger cerebral artery bursts its walls, often, according to Boyd, at the weak seam of a branching, and the patient dies from his apoplexy or survives to lead the vegetative life of a hemiplegic. And yet this fate cannot be upheld as a terror of hyperpiesia, for a similar effect from cerebral thrombosis is met with much more commonly in the aged of normal as well as abnormal pressure, though perhaps a half-decade later on the average. So that, all in all, the prognosis in a case of hyperpiesia is a most uncertain thing; as uncertain as that of a simple arteriosclerosis.

The chief factor in prognostic determination would seem to be the diastolic pressure, and after that the estimation of relative dominance in kidneys, heart and brain, with a knowledge of the average length of course of each type. The average durations of life in the different levels of diastolic pressure have been tabulated. They are shortened by the minority of catastrophic cases, whereas one can hope that the individual patient may, with fair luck and care, live to well on toward the upper limit, which exceeds the allotted three score years and ten. The only signs of a certainly brief course in the earlier years are the retinal changes, and the tokens in the urine and blood of a rapid kidney degeneration. Even anginal cardiac signs may allow of ten or more years of limited activity, although angina is a justifiably dreaded symptom. And one remembers that 44 per cent of hypertensives — almost half — take the slower, longer path of myocardial fatigue without pain.

TREATMENT

I will say little of treatment. The multiplicity of present remedies bespeaks their impotence. Fishberg states that simple hyperpiesics do best without treatment or knowledge of their malady.

Personally I have tried nitrites, iodides, the group of vasodilators which include euphyllin and metaphyllin, sodium thio-cyanate, and female sex hormone, and have found them all effective, with or without unpleasant by-effects, while the patient is resting. The patient returns to some activity and the pressure rises to former levels—perhaps fortunately, since these remedies are merely mopping up the leak, not stopping it, and possibly tend to upset a readjustment of the body to hypertension. Recently, a patient of mine, following avertin anæsthesia for extraction of teeth, dropped in her blood pressure from 200/115 to 95/62: it began to rise on the following day and after six days read 190/114. During the low interval she felt neither better nor worse than usual. The unpleasant symptoms which often accompany the use of thiocyanates are noted whether the blood-pressure is reduced or not; therefore they cannot be blamed on an enforced reduction of pressure.

Rest, physical and mental, lessens the body's demand for heightened pressure and seems rationally the most important treatment; it can be infused into a work-a-day life to grant prolongation of usefulness and comfort. The hypertensive is, by the same fact, usually an energetic and intelligent sort, and he can understand that he has allowed his self-driving to become a habit. With some guidance in what habits to watch, he can change them to new ones more of the tortoise than the hare, with often the achievement of the fable. Quiet work helps in preserving somatic and cardiac muscle tone and in promoting a restful contentment of mind; absolute idleness is damaging.

The normal blood-pressure rises as the day goes on. One may deduce that fatigue calls for production of more pressor substance to keep body and mind up to pitch—those pressors, whatever they are, that actuate hypertension. On this hypothesis, an hour or more's after-lunch relaxation, taken with strict immunity from telephone and door-bell, radio and family, is an excellent preventor of fatigue, and helps to cultivate the ability to relax. The English custom of afternoon tea is probably worth years of health to its habitués. Under the same principle, nerve sedatives such as bromide and small doses of phenobarbital are useful as a crutch at the start of treatment. Bed-rest I feel is indicated only in cases of threatened myocardial insufficiency, to allow the heart to overcome its fatigue. What use is it for hyperpiesia itself, when the temporary fall consistently disappears on resuming activity?

The best diet for the disease seems to be an evenly-balanced one of low enough value to keep down weight, and prohibition of large quantities of food or drink at one time. Nothing seems gained by reducing salt below the level of comfort, except in the presence of cardiac failure. It is curious to note how many of these patients are heavy salt-eaters. Very important it is that the patient be kept from developing a "phobia" which reacts directly on the hypertension itself.

Of late years the "blood-pressure" bogey has ousted the "leaky heart" bogey which kept its victim dying of a systolic murmur for seventy years or more. One hopes that the public never learn about diastolic pressure.

PROGNOSIS

Knowledge of hyperpiesia has not been common for more than fifteen years. That is too short a time to allow satisfactory knowledge of prognosis; many of our patients have outlived that period. Even the unfortunate with a diastolic pressure of over 130 mm., whom a few years ago we described to the relatives as of brief life here below, is still wielding a Hoover and defying all tables of averages. We have had knowledge of systolic high pressures of the so-called arteriosclerosic type for twenty-five years, and we know that a fair number of seventy-five-year-olds show it, although they are in normal health for their age. I see occasionally a very strong-minded old lady of eighty-nine whose pressure reads 250/82 mm., and whose doctor wrote me that twenty-five years ago she had a pressure of 180/ mm. Such instances allow one to pass on to the case in hand an optimism which does him physical good.

One cannot speak so hopefully of the true hyperpiesic, the case with diastolic raised above 100 mm., but at least it is known that the end is not certainly near, and may be far. White mentions serious prognostic signs as being a diastolic pressure over 130 mm., marked retinal changes, pulsus alternans, gallop rhythm and cardiac asthma. Fortunately most cases show no marked retinal changes, and these cardiac signs are often postponed into late life, so that White's context is comforting— "a serious prognosis should not be given on the finding of hypertension or slight cardiac enlargement alone." This is encouragement to be passed on, and so is the finding that pressure does not increase with age and may even recede. One recollects that nearly half of the cases take the path of myocardial insufficiency without angina, and that one is accustomed to see the end of this in old age. There are, then, good reasons why the patient can be relieved of some of the dread of "high blood-pressure" without undue strain on the doctor's conscience, and I, personally, do not feel that one is fitted to give a prognosis in the average case until he has observed it surreptitiously for a year or more.

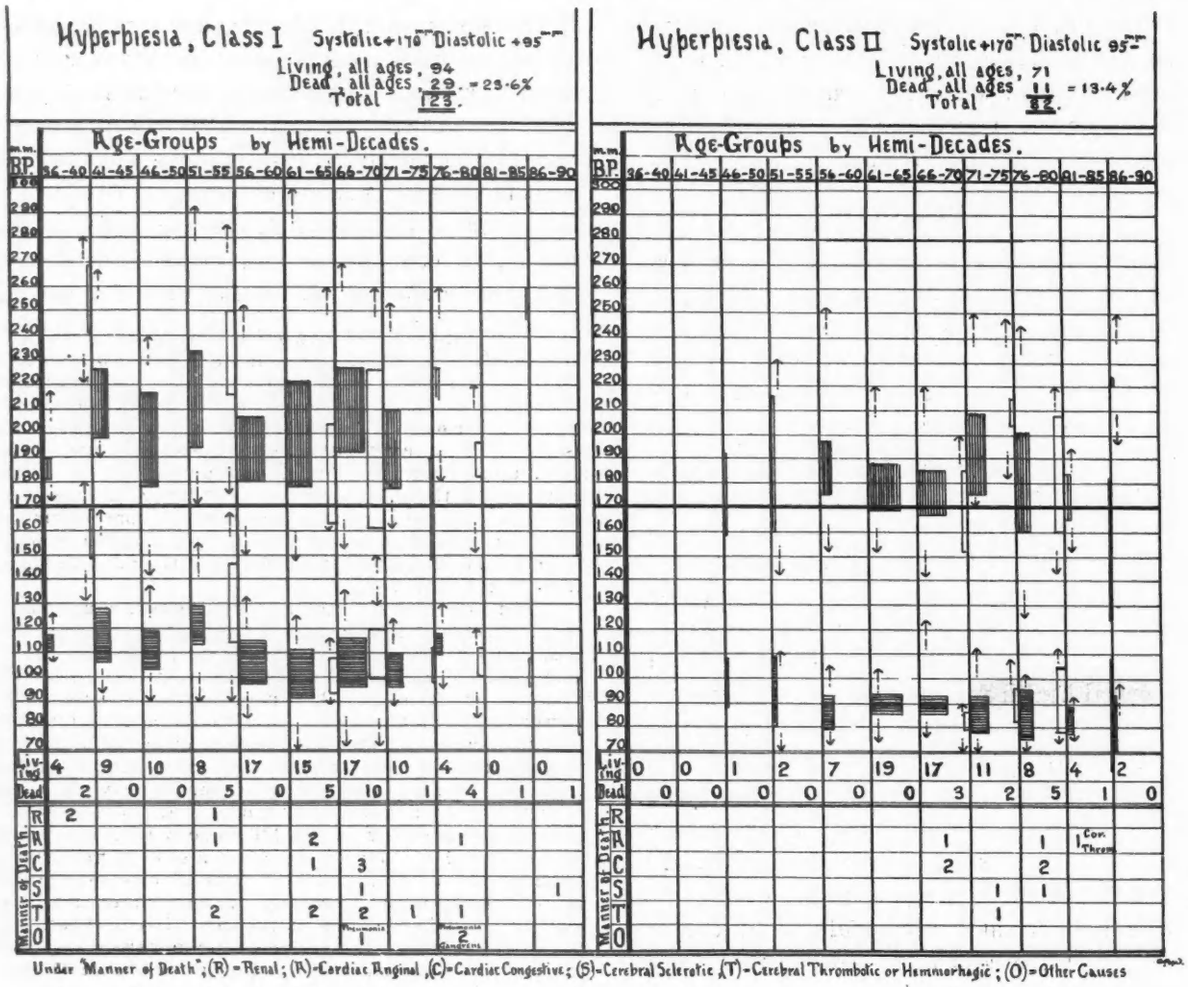
The notes so far have been a composite of gleanings from authoritative literature and of my own personal experiences. As an illustration I have attempted an analysis of my own small series of hypertensive cases. The collection has been made from private cases because they permitted more chance of follow-up; and for the tedium of compilation and detail I am much indebted to Miss M. Deakin, my office nurse.

Two hundred and five case records were studied: 165 of patients still living, and 40 of patients who had died since coming under observation.. To illustrate distinctions between two apparent types of hypertension, the cases were divided into two main classes; those with an average diastolic pressure of over 95 mm., and those of 95 mm. and under. It is to be emphasized that the average height for each case is the one referred to; many cases with average normal diastolic cross and recross the 95 mm. dividing line on repeated readings, although rarely do they depart more than 15 mm. from it.

The choice of a systolic figure to use as a minimum for hyperpiesic tables was difficult, and in this the unfolding of the two main types gave one some help. The more tolerant of the authorities rule that a systolic pressure much above 160 mm. must be considered hypertensive at any age. In my case records I noted many instances of systolic pressure over 140 mm., the perfect normal, but found that until the systolic reached 170 mm., there was no tendency toward an accompanying abnormal diastolic. There were two exceptions—systolics in the 140-60 zone with diastolics up to about 110. One of these was a case of pernicious anæmia; the other was one of Graves' disease relieved by thyroidectomy; therefore, there was chance for ambiguity in both. But at the zone 170 to 179 mm., systolic, the diastolic accompaniments began to show readings ranging from 90 to 102, with one exception reaching 126 mm.; and once above 180 mm. systolic, there seemed to be no parallelism between diastolic and systolic; very high diastolics might show at once. The zone about the 170 mm. systolic pressure mark seemed to be a natural division line on the basis of diastolic accompaniments; therefore it was chosen as a systolic minimum for the series of hypertension cases. The 123 cases with average abnormal diastolics were placed in Class I; the 82 with average normal diastolics were placed in Class II; and the age-incidence and death-incidence of each class have been condensed into graphs. In the two graphs, rectangles show average blood-pressure readings of cases in each age-group (hemi-decade). To obtain figures as a basis for the rectangles an average was struck of the multiple readings of each case, and an average of these individual averages was used for the graph of the particular age-group. The height of each rectangle shows the average variation of pressure, and the width is proportionate to the number of cases in the age-group. Vertical hatching denotes systolic readings; horizontal hatching, the diastolic readings; and the vertical arrows indicate extreme variations of pressures in each age-group. Blank rectangles are of deaths.

There are points of similarity in the two graphs, but enough points of dissimilarity to make one feel that they show two distinct types.

The similarities are that after the age of 45, neither shows a tendency to progressive rises of pressure or a fall with old age, that there is no consistent difference either in average


Hyperpiesia, Class I Systolic +170 Diastolic +95
Living, all ages, 94
Dead, all ages, 29. = 23·6%
Total 123.
Age-Groups by Hemi-Decades.
Hyperpiesia, Class II Systolic +170 Diastolic 95
Living, all ages, 71
Dead, all ages 11 = 13·4%
Total 82.
Age-Groups by Hemi-Decades.
Under "Manner of Death"; (R) = Renal; (A) = Cardiac Anginal; (C) = Cardiac Congestive; (S) = Cerebral Sclerotic; (T) = Cerebral Thrombotic or Hemmorhagic; (O) = Other Causes

pressures or in extreme pressures or in variation of pressures between the living and deaths in each age-group, and that the modes of death, renal excepted, are similar.

The dissimilarities are more numerous. In the high-diastolic class the upper average systolic reading jumps from 190 mm. at 36 to 40 years to 226 mm. at 41 to 45 years, and thereafter forms an irregular plateau between the 207 and 234 mm. levels up to the age of 76 to 80. Several extreme readings approach the 300 mark. The upper average diastolic reading varies from 108 to 129 throughout, and diastolic rectangles shift, in all instances but two, with their systolic mates. There are two deaths in the 36 to 40 group; both of these are of the renal or malignant type. Thereafter, deaths begin again in the 51 to 55 year group. The thinning-out process becomes marked in the 76 to 80 group and no patients over 80 are still living.

In the normal diastolic class there are no cases under 46 years of age, and the systolic plateau varies between the 184 and 209 mm. levels up to 81 to 85 years. Two cases surviving to the 86 to 90 year group display a height of 223 mm., but, excluding these, the systolic level is definitely lower in this class. In general, too, the variation is lower, and the extremes do not go much over 250 mm. A striking feature is that deaths do not begin until the 66 to 70 age, whereas in the high-diastolic class, even after a hiatus following the early renal deaths, they begin again at 51 to 55 years. The thinning out is shown at 81 to 85 years, but two patients are still living in the 86 to 90 year group. This longer expectancy of life alone is enough to lead one to the impression that there are two definite types of hypertension, and that the diastolic pressure is the deciding criterion between them and between a doubtful and a cheerful prognosis.

The phenomenon of fall in pressure with age is one which I have met only once. In several cases it seemed apparent, but further readings dispelled the illusion, as they did the illusion

of permanent benefit from drug therapy. Nor have I seen blood-pressure fall with cardiac failure, except under two circumstances; one as a terminal sign in either congestive or anginal failure, and the other sometime in the first four days following acute coronary occlusion. In the latter contingency the pressure has usually risen to somewhere near its former level within a few months as recovery progresses. Indeed, it is remarkable how pressure keeps up in the presence of a heart distressed to the extent of pulmonary congestion and cyanosis.

Histories of known hypertension were not common, being obtained only in 49 cases. In Class I, 8 out of 23 cases under 50 years of age gave histories, one dating known high pressure back to the age of 31 years, others to about 40. One active patient of 70 years, with a pressure of 232/108, had known of hypertension for twenty years. In Class II, no cases under 56 years of age gave histories of hypertension. One patient of 72 stated that her blood-pressure had been over 200/ for 15 years, and another now 89 years old had a written record of a pressure of 180/80 twenty-five years ago.

Symptoms and signs of the commoner sort only were tabulated — atheroma, cardiac enlargement, alveolar moisture, dyspnœa, angina and congestive failure pertaining to the cardiac "path"; and dizziness, headaches or paræsthesias, cerebral sclerosis, and cerebral vascular accidents, pertaining to the cerebral "path". It would be too tedious to quote exact figures of distribution, but some generalities of interest evolved from compilation. Referring first to Class I, one finds a group of patients of under 55 years with blood pressures of over 210/120 who are apparently in perfectly robust health, with no symptoms. Some of these have known of hypertension for years; one, aged 43 years, for 12 years. Dizziness and dyspnœa on exertion are the predominant symptoms of the remainder. In many cases both are of slight degree, and the vertigo is an intermittent thing, occurring perhaps at months' intervals, often accompanied by nausea or even vomiting, but not of the intensity of a Ménière's syndrome nor added to deafness and tinnitus. Dyspnœa is difficult to assess in the older patients, since it is often instinctively or purposely avoided, especially by anginal patients, and then honestly but thoughtlessly denied. The incidence of both symptoms becomes greater after 56 years, and thereafter increases very little. Both are definitely more common, age for age, in the raised diastolic class, but since they are almost absent in the 36 to 40 year group, one would deduce that they have been caused by sclerosis of arteries and myocardium which has been advanced five years or more by hyperpiesia.

Cerebral sclerotic signs begin at 51 to 55 years with a case of Ménière's syndrome, and thereafter one or two cases of some form of cerebral arterial sclerosis are seen in each hemidecade. Cerebral vascular accidents begin in the 51 to 55 year group. In Class II the anginal syndrome does not appear until 56, but in the next ten years the proportion is larger than in the high-diastolic group. Congestive heart failures are rare, and do not appear at all until after seventy. It would appear that the height of systolic pressure in Class II cases is no index of the severity of symptoms.

Urinary findings in the great majority of cases were entirely negative in both classes; in a little over 20 per cent of Class I there was found a faint trace of albumin with a few casts, but Mosenthal or Volhard tests were normal. This is, of course, excepting the three "renal" cases. Of the Class II cases, about 9 per cent showed equivalent urinary findings. Of 47 eye-grounds examined in the Class I division, 14 seemed normal; the remainder showed merely the dilated veins and sometimes notching and widened arterial reflexes without any papill-œdema. Again, this excepts the renal cases which showed marked retinopathy. Of 18 fundi of Class II, 5 seemed normal; the remainder showed signs as in Class I.

An attempt was made to estimate the state of health of the different age-groups. Most of the patients are still within my ken, but enough have been lost track of to make the estimate uncertain, and a few were seen only once or twice. But I can safely say that 21 out of the 31 Class I patients under 56 years of age are leading active lives, and only 3 are much restricted in activity, one being a diabetic and another a victim of myxœdema. From 56 to 60, only 11 out of 17 are active or fairly active, and after that the number of semi-invalids and invalids steadily increases. In Class II, 30 out of 36 cases in the 60 to 70 year groups are still

fully active, so that there is a distinct difference in this respect between the two classes.

To compare the fate of elderly patients of the hypertensive series with that of their eutensive contemporaries, records were studied of 159 other patients of over 60 years of age with systolic pressures under 170 mm. One hundred and four of these were living; their average blood pressure was 144/75, and showed no tendency to increase with age-groups. Four of these patients, all with systolic pressures under 140 mm., suffered from anginal syndrome. Tabulated, the comparison reads as follows; it is broad, but illuminating:

Hypertensives (with increased diastolic) over 60 years, 68. Deaths 22. Mortality 32.3 per cent.

Hypertensives (with normal diastolic) over 60 years, 72. Deaths 11. Mortality 15.3 per cent.

Normal tensives over 60 years, 159. Deaths 55. Mortality 34.6 per cent, of which deaths from sclerotic cardiovascular disease made up 13.2 per cent.

SUMMARY

1. The etiology of hyperpiesia, beyond the recognition of arteriolar spasm, is hypothetical.

2. The prognosis of hyperpiesia is incompletely known because of, as yet, too short a period of observation.

3. An analysis of a series of 205 case records shows some reason for separating cases of raised blood-pressure into two classes—those with raised average diastolic pressure (Class I), and those with normal average diastolic (Class II)—and for naming 170 mm. systolic pressure as an approach to a natural rather than an arbitrary minimal reading for true hyperpiesia.

4. It would be desirable to limit the name "hyperpiesia" to those cases with a raised diastolic pressure (which occurs, with rare exceptions, only when the systolic pressure remains over 170 mm.), and to clearly distinguish by another name the hypertensive condition whose criterion is a normal diastolic pressure and whose course is at least not far from benign. One might suggest the designation "hyper-systolic tension".

5. Little has been observed of the mode of onset of hyperpiesia, but it would appear not to be of gradual development; rather, of devolopment within five years and thereafter maintenance of a roughly level pressure. This is borne out both by individual records and by comparison of age-groups.

6. It is suggested that the phase of rising pressure in Class I takes place before the age of 45, and in Class II mainly before the age of 55.

It is now possible for a hospital patient to lie in bed and push a nurse's call button, in response to which the nurse's voice immediately answers through a loud speaker, "What is it please?" The patient, without moving, may answer in the lowest tone which could be heard if the nurse were in the room, "A drink of water please." The nurse replies through the loud speaker, "Right away." This saves one-half of the nurse's time as required by the old method. If the patient is convalescing and wants to hear the ball game, horse race, political speech, soft music or songs, the patient pushes the nurse's call button and the nurse's voice through the loud speaker says, "What is it please?" and the patient asks for a favourite radio station which is immediately connected with the loud speaker in the patient's room. The system may be used for sound reinforcement in the chapel with a microphone and loud speakers, and chapel services may be distributed to patients' rooms the same as radio programs. On Sundays the matter of keeping down the number of visitors is quite a problem for the hospital superintendent. By this new system the superintendent announces to all rooms, "Five minutes until closing time. All visitors will kindly leave." Or the floor nurse may announce in any particular patient's room, "Please be quiet. The patient in the next room is being disturbed." (The Holtzer-Cabot Electric Company of Boston are the manufacturers.)

PROGNOSIS IN PULMONARY TUBERCULOSIS. — The value of radiology in the prognosis of pulmonary tuberculosis is described by F. B. Trudeau, who states that the death rate is in direct proportion to the amount of lung involvement. The prognosis in the exudative type of disease is decidedly more unfavourable than in the proliferative type, and the presence of cavities nearly doubles the probability of death within five years. Cavity cases showing improvement under treatment have approximately five times as favourable a degree of prognosis as those in which the cavities become larger during sanatorium residence. Patients in whom repeated x-ray examinations are constantly favourable have more than twice as good a chance of being well at the end of five years, and only one-fourth as great a chance of being dead, as those who have increased lung shadows. Such increase suggests a prognosis about equally as unfavourable as that indicated by the presence of pyrexia. Patients with both pyrexia and increased lung shadows have six times as unfavourable an outlook as those who are free from fever and whose x-ray examinations show constant improvement. Increased comparative x-ray shadows are of much graver prognostic significance than such increased physical signs as râles. The yearly follow-up records of 600 patients at the Trudeau Sanatorium indicate that the relation of "well," "relapsed but now well," "chronic," and "dead" is in direct ratio to the incidence of x-ray increase while they were having institutional treatment.—*J. Am. M. Ass.*, 1936, **106:** 592. Abs. in *Brit. M. J.*

MENTAL DISEASE FOLLOWING CARBON MONOXIDE POISONING*

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THE deadly qualities of carbon monoxide have been recognized since antiquity. Hannibal used it to destroy many of his captives, and Seneca utilized it as a means to suicide. Troja is supposed to have been the first to make a definite scientific investigation of the question, when in 1778 he described the cherry-red colour of the blood. Since that time many monographs and reviews have been written setting forth the various symptoms due to carbon monoxide poisoning. These symptoms are multiple and diverse, and have been described as initiating such conditions as hemiplegia, amnesia, localized paralyses, multiple neuritis, paralysis agitans, disseminated sclerosis and complete dementia. It has only been of recent years that the mental aspects of these cases have been recognized. It is true that headache has been a prominent symptom in most cases, and Aristotle observed that men suffered from "heaviness of the head" when exposed to coal gas, but the idea that insanity might be a result seems of fairly recent origin.

During the past few months we have admitted to the Verdun Protestant Hospital two patients who, prior to exposure to carbon monoxide, were apparently normal, but who after the exposure developed grave mental disorders. It was not possible to obtain spectroscopic analysis of the blood in our cases. It should be noted that Pouchet[1] and later workers have shown that carbon monoxide hæmoglobin is detectable in the blood for many days after exposure. Further, in the early stages, when the concentration is comparatively high, several simple presumptive tests are possible. These simple procedures can also be applied to test the suspected atmosphere.[10]

CASE 1

(No. 9284) A.J.M., aged 19 years, a nursemaid by occupation, was exposed to carbon monoxide in the latter part of January, 1935, from an automobile exhaust. The patient, with two other women, was going home with three male companions in a motor car. The men sat in the front seat with a window open. The carburetor and exhaust were known to be defective, and the exhaust gas in the car was objectionable, but it was considered too cold a night to open any other window. They were in the car altogether about one hour. One of the women became unconscious and had to be taken from the car; the other collapsed after she reached home. In view of the rapid onset of symptoms in these two women, one assumes that the third and most seriously affected was exposed to a relatively high concentration of carbon monoxide. Relatives state that on reaching home, the girl appeared nervous and her eyes seemed to stare. She claimed she did not feel well, and that food, even a cup of coffee, made her nauseated. She remained ill for several days, and a physician told her family that she had influenza with pleurisy. One week after the exposure she began to talk peculiarly, having delusions of reference and that her food had been poisoned. She was described as being catatonic, with hallucinations of sight and hearing. She became violent toward her father and mother, and finally refused to take her food. On admission to hospital, one week after the onset of symptoms, she was resistive, refused her food, and did not comprehend or notice anything except that she would curl up, whimper and cry occasionally when approached. Delusions of reference, and auditory as well as visual hallucinations were marked. It was necessary to provide nourishment by spoon feeding. Her attempts at speech were slow, low-pitched and incoherent. Physical examination showed an underweight juvenile dysplastic, with evidence of recent malnutrition. There was retention of urine. Her face and neck showed a considerable degree of acne. Her eyes were constantly in motion, glancing from side to side. There was no disturbance of conjugate movement, and no nystagmus. Her fundi were normal except for a fine visible thread extending from the fundus of the right eye to the posterior surface of the lens, probably a remnant of the hyaloid canal. There was marked hippus of the pupils. The tendon reflexes were all exaggerated. At rest the muscles were soft and passive, but when movement was first attempted rigidity set in. This was followed by cogwheel rigidity when elbow- and knee-flexion were attempted. This disappeared after several minutes of passive movement, but would return later. The corneal reflex was present; Hoffman's sign was negative; the abdominal reflexes could not be elicited; the plantar showed flexion on both sides. Although the patient had sufficient bed clothes she showed a marked pallor, with pilo-motor reflex. There was no *tache cerebrale*. Flexibilitas cerea was present, but not constant nor sustained; a hand placed lightly on her shoulder caused her to sink slowly nearly to the floor. The respiratory system was normal at the time of this examination.

After two weeks the patient began to eat voluntarily. Once or twice she joined in a limited conversation with the examiner, but would not answer questions. She whimpered a good deal but did not actually weep. She began to improve gradually. A month after admission she was up and about, appeared rather helpless and confused, but made low-voiced, often incoherent attempts at speech, and was heard to say "They are over there". Three months after admission she ate and slept well, was less apprehensive, and her general nutrition had improved. Four months after admission improvement was maintained, but auditory hallucinations were demonstrated and they appeared to be the cause of a remaining

* Read before the Psychiatric Section of Montreal Medico-Chirurgical Society on April 30, 1935.

tendency in the patient to be apprehensive at times. She was then attending occupational therapy classes. During the fifth month these hallucinations became less troublesome, and, in fact, she said some of them were enjoyable but she could not describe what the voices were saying, although she recognized the voice of her mother. Memory for events prior to and just following admission was demonstrated. Improvement during the seventh month was most noticeable. Hallucinations of hearing (vague voices) became infrequent; insight appeared good; memory restoration was adequate; emotional range was full and syntonic; conduct and attitude were normal.

Physical examination demonstrated some retinal atrophy about the optic discs; marked increase in the biceps, supinator and knee jerks, the right side being extremely active; a gain in weight of twenty pounds since admission; and normal blood pressure; but no other features of note.

The patient was recommended for trial at home with follow-up observation.

CASE 2

(No. 9220) M.K., aged 66, a junk dealer, was born in Poland and had been in Canada for thirty-one years. There was no relevant family history and no known mental disease prior to this attack. He was admitted to hospital on January 5, 1935, with the history that he had been unconscious from coal gas poisoning, four weeks before, for a period of thirty-five hours. His wife died in the Royal Victoria Hospital on December 5, 1934, from bronchopneumonia following the same carbon monoxide exposure.

On admission the patient was quarrelsome, confused, resisted dressing, feeding, examination and care of his person. He lay like a log for several days if undisturbed. If one persisted in these attentions he became violent. He occasionally repeated words but would not answer questions. His speech was slurring and there was incontinence.

Physical examination showed a noticeable pallor of the mucous membranes and marked œdema of the lungs. An extreme degree of cogwheel rigidity was present on flexion of the elbows and knees. The tendon reflexes were markedly hyperactive. Shortly after his admission the œdema of the lungs increased, the temperature rose to 103° F. and he was desperately ill for a few days. Tube feedings were necessary. The blood Wassermann and cerebrospinal fluid examinations were normal, in spite of the resemblance clinically to general paresis, and in the face of a provocative dose of tryparsamide. After one month the patient began to speak more clearly, but the reflexes were still hyperactive, and cogwheel rigidity was even more marked than before. A definite intention tremor was present. The lower extremities gradually regained their strength. After one and a half months he was able to walk, although he was unsteady on his feet. His speech improved gradually, as did his conduct. He could at this time remember nothing after the middle of November, 1934, (that is from a few days prior to the exposure to carbon monoxide) although his memory was intact for earlier events. The tendon jerks were still hyperactive and cogwheel rigidity was still marked.

On March 10, 1935, two months after admission and three months from the date of exposure, the patient was allowed out on trial. Amnesia for the above-mentioned period had practically disappeared, although there were still a few details he could not remember. The tendon-jerks were less active and cogwheel rigidity was slight. There was no disturbance of speech, coordination or gait. On April 10, 1935, four months after exposure, he was able to recount in detail all the events up to the time of and following the accident, except for the thirty-six hours when he was unconscious and for a period of a few days just before and following his admission to hospital. He was quiet, cooperative and was considered physically and mentally asymptomatic on discharge.

The first case on casual examination resembled a hebephrenic schizophrenia in a juvenile type. In favour of this it might be noted that the girl was the only poorly developed member of the family and her home life had been unhealthy, the mother being a confirmed troublesome alcoholic. However, the definite history of exposure, the symptoms presented that are foreign to the schizophrenic syndrome, and the gradual and continuous improvement are in favour of a temporary organic change.

The second case resembled general paresis. This similarity between carbon monoxide poisoning and paresis has been noted in other hospitals. August Meyer[3] reported a case in 1930 of pseudoparesis resulting from carbon monoxide poisoning. Moreover, this case is interesting in that there was a fairly long period following exposure when the man was apparently free from symptoms save for a headache. According to Sibelius and others[4] this is fairly common in old age as here we have the toxic changes of the carbon monoxide added to an already deteriorated vascular system. According to Bleuler[5] recovery at this age is uncommon,—"If the patients do not recover entirely, then the organic symptom complex, accompanied usually by conspicuous severe memory defect and depression, remains as an expression of the diffuse destruction of the brain tissue. Between the first toxic sleep and the later development of the symptoms several days or even weeks free from disturbance may intervene." Kraepelin also emphasizes this latent period between the exposure and the development of severe symptoms.

McConnell and Spiller[6] have described three types of carbon monoxide poisoning—acute, chronic and relapsing. Those suffering from the acute type either die immediately or at once pass into such states as pneumonia, Parkinsonism, mental deterioration, etc. The chronic sufferers are usually garage men who exhibit headache, vertigo and memory defects. These cases are far more common than is realized, since inhaled air containing 0.15 per cent of carbon monoxide will produce headache. McNally[7] examined the blood of 40 workers from three different garages and found that 69.5 per cent of them showed carbon monoxide hæmoglobin. The relapsing type of case is well

illustrated by patient M.K. The sequelæ here cannot be due to the presence of the gas itself, but must result from the injury making itself manifest at some later date. Hill and Semerak[8] and many others have found that the damage was usually most marked in the globus pallidus. The amount of damage depended on the age of the patient, the quality of gas inhaled, the duration of life after the accident, the amount of degeneration of the vascular system, and pre-existing disease of the brain. There is however no typical carbon monoxide brain and the picture is frequently most complex. For instance, such changes have been found as degenerative and atrophic conditions in the ependymal cells of the choroid plexus; there may be swellings of some cells and shrinkage of others; minute hæmorrhages may occur throughout the brain; there may be demyelinization of the white matter; or softening of the cortex may appear, especially in the infragranular layer; the olfactory system may be affected; even the anterior horn cells of the cord may be damaged. Thus it is seen that an endless variety of nervous and mental symptoms may develop following carbon monoxide poisoning. In fact those cases reported in the literature are usually similar in some respects but vastly different in others. In 1932 R. P. Mackay[9] reported a case which showed typical Parkinsonism, due undoubtedly to encephalitis following exposure to carbon monoxide. Wilson and Winkleman[10] reported a case in 1925 showing multiple neuritis and cerebral involvement, which at autopsy revealed a zone of necrobiosis involving part of the third and the entire fourth cortical layers (Brodmann's classification). These layers have been observed to be susceptible to other intoxications.

Since accidents from exposure to carbon monoxide are becoming more common, it is reasonable to suppose that an increasing proportion of nervous and mental disease will be brought to our attention from this source. Hill and Semerak (*loc. cit.*) after examining the brains of 32 patients who died from such accidents were struck by the amount of degeneration in the lenticular nucleus, and concluded that any individual with a lesion here should at least be suspected of suffering from chronic monoxide poisoning. One would point out the frequency of symptoms referable to congestion of the lungs or pulmonary œdema.

The two relapsing cases here reported exhibited what might be described as the *carbon monoxide syndrome.* This comprises a history of exposure, pulmonary œdema, a latent period, clinical evidence of lenticular nucleus involvement, and the common features of an organic psychosis with amnesia, confusion, and emotional instability. The survival and apparent complete recovery of these patients is unusual.

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EFFECT OF CAROTENE AND VITAMIN A ON PATIENTS WITH DIABETES MELLITUS: EFFECT OF DAILY ADMINISTRATION OF CAROTENE ON BLOOD CAROTENE OF NORMAL AND DIABETIC INDIVIDUALS.—E. P. Ralli, A. C. Pariente, H. Brandaleone and S. Davidson observed the effect of the continued daily oral administration of 0.3 per cent carotene in oil on the blood carotene and cholesterol of a group of four normal and four diabetic persons. Following the administration of 1 c.c. of the carotene solution to three normal subjects and to three diabetic patients there was a greater increase in the blood carotene in the latter and a more gradual return to the fasting level after carotene was stopped. Following the administration of 5 c.c. daily to one diabetic patient and to a normal subject whose blood carotene level had been elevated as a result of the previous administration of a large dose of carotene there was a still greater increase in the blood carotene in the diabetic patient above that of the normal subject and clinical evidence of carotenæmia appeared. The blood cholesterol was estimated in three normal subjects and three diabetic patients. This rose with the blood carotene in one diabetic patient and in two of the normal subjects. To explain this increase in blood carotene in diabetic patients, both before and after carotene administration, it is suggested that in the diabetic patients the ability of the liver to convert carotene to vitamin A is diminished, and that this results in an increased concentration of carotene in the liver, which interferes with the absorption of carotene from the blood.—*J. Am. M. Ass.*, 1936, **106:** 1975.

THE ASSOCIATION OF PREGNANCY, HYPOCHROMIC ANÆMIA AND ACHLORHYDRIA*

A PRELIMINARY REPORT

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THE association of these three conditions is an extremely common one. Their interdependence is indisputable. What the underlying initial cause may be is quite obscure, and also why pregnancy should be an etiological factor. Hypochromic anæmia is rarely found in nulliparous women. Therefore there must be an etiological factor in pregnancy, and as pregnancy produces its most profound changes in the endocrine system—exclusive of the uterus and its contents—for these and other reasons the inference is at least suggested that the primary cause is to be found in an endocrine dysfunction.

HISTORICAL

A great deal of study has been devoted to hypochromic anæmia with achlorhydria in the last few years. This condition has recently been described under numerous synonyms. These are microcytic anæmia (Faber[1]), idiopathic hypochromic anæmia (Mills[2]), chronic chlorosis (Mettier and Minot[3]), primary hypochromic anæmia (Dameshek[4]), hypochromic anæmia with achlorhydria (Waugh[5]), and others. In spite of all the researches the etiology is still obscure. The terms "idiopathic" and "primary" point clearly to an unknown origin. However, it is now definitely known that it is a disease confined almost exclusively to women of about middle age, and is characterized by pallor, weakness, atrophied tongue, brittle finger nails, achlorhydria and hypochromic anæmia, the chief determining factors of which are a lower hæmoglobin content, without a corresponding diminution in red blood cells. The onset of the disease is always insidious. In Wintrobe and Beebe's series[6] the duration varied from 2 to 22 years with an average of about 8 years. Many factors, such as surgical procedures and infectious diseases have been enumerated as etiological, but pregnancy, though not the primary, is the outstanding, common intermediate cause. In about 20 per cent of cases the onset can be definitely associated with pregnancy.

METHODS

This work was begun primarily to determine the blood changes incident to the influences of pregnancy upon the hæmopoietic structures of the body, and to determine at what stage of the pregnancy these influences were most marked. Incidentally, attention became drawn to the subject of hypochromic anæmia, and a study of pregnancy influences upon its etiology and degree. This series comprises 200 consecutive, non-selected, cases of pregnancy. In all of these a complete cytological blood examination and hæmoglobin estimation were made; and in 115 of these analyses were undertaken of the gastric content during starvation and at one hour after ingestion of toast and tea. Only average figures will be given here, leaving the details for a later report.

The findings have been compiled from two points of view: (1) from the duration in months of the pregnancy under investigation, and (2) from the number of pregnancies that have preceded the present one. We have therefore divided the 200 patients into (I) pregnancy trimesters groups, and (II) into (*a*) primiparæ, and (*b*) multiparæ who have had a considerable

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This research was made possible by a grant from the Banting Research Foundation and from other sources.

interval between their successive pregnancies, and (*c*) multiparæ who have had a rapid series of conceptions.

Hæmoglobin.—The general average amount of hæmoglobin was 75 per cent = 10.50 g. During the first and second trimester the reduction in the hæmoglobin was less marked than during the third trimester. The averages are given in Table I. First trimester, 80 per cent = 11.20 g.; second trimester, 75 per cent = 10.50 g.; third trimester, 65 per cent = 9.10 g. On the other hand, the averages in group II, (*a*), (*b*), and (*c*) are strikingly eloquent (see Table II). Multiparous women who had had pregnancies in rapid succession show a great drop in hæmoglobin cellular content.

Gastric analyses.—Analyses were made upon 115 cases, 60 ante- and 55 post-partum. In the first trimester, 51 per cent of the cases showed normal gastric acidity; 40 per cent had hypochlorhydria, and 9 per cent achloryhdria. In the second trimester, 13 per cent showed hypochlorhydria and 87 per cent showed achlorhydria; in the third trimester, 0.5 per cent showed normal acidity, 3.5 per cent hypochlorhydria, and 96 per cent achlorhydria.

TABLE I

TABLE OF AVERAGES IN FIRST, SECOND AND THIRD TRIMESTERS OF PREGNANCY

Group I	*Average hæmoglobin per cent and gramme*	*Average number of red blood cells in thousands*	*Average colour index*	*Average reticulocytes per cent*
First trimester	80% = 11.20 g.	4,300	0.93	2.0
Second trimester	75% = 10.50 g.	4,100	0.91	3.0
Third trimester	65% = 9.10 g.	3,700	0.87	5.0

Degree of post-partum recovery.—In 85 per cent of the foregoing cases submitted to post-partum analysis acidity began to return within a week after delivery. Although the free hydrochloric acid was far from the normal quantity, goodly quantities were present. In 12 per cent there was a mere trace in the same post-partum interval, and in 3 per cent there was a persistence of achlorhydria. The 12 per cent of the cases with a mere trace of free acidity and the 3 per cent of cases with persistent achlorhydria all belonged to the group II (*c*), that is, multiparous patients who had a rapid succession of pregnancies. Only a few cases have so far been examined one month *post partum*, and in 2 per cent of these, achlorhydria still persisted.

TABLE II

TABLE OF AVERAGES IN PRIMIPARÆ, MULTIPARÆ WITH DISTANTLY SPACED PREGNANCIES, AND MULTIPARÆ WITH RAPIDLY SUCCEEDING PREGNANCIES

Group II	*Average hæmoglobin per cent and gramme*	*Average number of red blood cells in thousands*	*Average colour index*	*Average reticulocytes per cent*
Primiparæ	79% = 11.00 g.	4,200	0.94	2.5
Multiparæ with distanced pregnancies .	79% = 11.00 g.	4,250	0.94	3.0
Multiparæ with rapidly succeeding pregnancies	58% = 8.12 g.	3,600	0.80	6.0

Erythrocytes.—The red blood cells were also reduced in all cases, but much less so than the hæmoglobin.

The average count in the 200 cases was 4,100,000.

Group I.—First trimester average, 4,300,000; second trimester average, 4,100,000; third trimester average, 3,700,000.

Group II.—(*a*) Primiparæ showed an average of 4,200,000; (*b*) multiparæ with well spaced pregnancies, 4,250,000; (*c*) multiparæ with rapidly succeeding pregnancies, 3,600,000.

In the last group reticulocytes were found in greater number than normal, and the increase was proportionate to the degree of anæmia. Microcytes followed this same rule fairly closely. This is more clearly brought out in the colour index, which becomes lower, the greater the anæmia.

DISCUSSION

We have found that during pregnancy there is a definite tendency to the development of a progressive hypochromic anæmia; that this anomaly becomes most pronounced in cases where pregnancies succeed one another in rapid

order. There is also a marked diminution, or complete disappearance, of free hydrochloric acid in the later months of gestation, and a strong tendency to a return to the normal, in the milder cases, in the early days *post partum*. This return to the normal is delayed temporarily or permanently in parous women with rapidly succeeding gestations.

What explanation can be offered for the mechanism which produces the anæmia and associated gastric dysfunction? Normal erythropoiesis requires several coordinated factors to create a normal cellular blood balance; first, a normal myeloid system; second, a sufficiency of the constituents of normal erythrocytes and hæmoglobin in the body; and third, a normal gastric factor. We can assume that the average pregnant woman has a normal myeloid system which should be capable of responding to a very considerable increase, if needed. The hypochromic type of anæmia shows that in all probability we are dealing with an iron deficiency, and the hypo- or a-chlorhydria suggests a gastric factor lacking the necessary stimulating influence to blood production.

Why should pregnancy lead to a disturbance of these two factors? Since hypochromic anæmia is a disturbance limited almost exclusively to women, and particularly developing during pregnancy, and since, where not unduly prolonged or frequently repeated at short intervals, it tends to disappear rapidly with the end of gestation, we are justified in the inference that it receives its chief stimulus from pregnancy, and, more particularly, from the uterine content. Therefore, the fetus, or its appendages, or both, are in all probability the determining factors in this disease. The fetus not only develops its own blood supply during the intrauterine life, but actually develops a polycythæmia of high grade during its prenatal stage, to overcome the normal degree of anoxæmia in which it lives before birth (Goldbloom and Gottlieb[7]). This normal anoxæmia is due to imperfect exchange of gases between maternal and fetal bloods, as compared with that between air and blood in lung aeration. The extraordinary fetal hæmopoiesis requires the same factors as did the same process in the mother. We can assume a normal myeloid system in the normal fetus. Iron, however, must obviously be supplied from the outside, so the fetus draws upon the mother's supply, as it does in the case of calcium and other requisites. Further, there is no gastric secretion in the fetus to supply the stimulus to the myeloid system, and free hydrochloric acid does not appear in the stomach of the infant until some time after birth. The only evidence we have of the presence, or absence, of the gastric factor is the presence, or absence, of free hydrochloric acid in the stomach. It is tempting to assume, therefore, that the fetus draws upon the maternal gastric factor as it does upon her supply of iron. This explains, in our opinion, the development of hypo- or a-chlorhydria, and hypochromic anæmia, in pregnancy, and that these stand in the relation of cause and effect. It also offers an explanation for the increasing degree and persistence of these diseased states, with the increase in number and rapidity of successive pregnancies, which lead to permanent functional dystrophy through exhaustion of maternal gastric reserve.

What is then the relation of these cases of hypochromic anæmia and the smaller number of cases in which pregnancy did not enter as a causative agent? A definite connection can probably be established. However, the majority of cases of hypochromic anæmia are in parous women. The small minority in nulliparous women or girls, and the very occasional case in the male (so occasional as to be a curiosity) can naturally occur when these disturbances in the body iron content, and gastric factor, may be the initiating cause.

This leads us to the question of the condition being a separate disease entity. The appearance of the disease chiefly in parous women, but also, though much more rarely, in nulliparous, and the very occasional case in men, disproves, in our opinion, a uniform etiology. The anæmia must necessarily be secondary to something that interferes with iron metabolism and the gastric factor. Therefore, such terms as "idiopathic", or "primary", seem to be out of place in connection with this type of anæmia.

It was stated in the first paragraph that, as pregnancy produces its profoundest changes in the endocrine systems, it is suggested that the primary cause of hypochromic anæmia and its associated lesions is to be found in the endocrine system. Also there is another reason. Every function of the body owes its normal continu-

ance to the operation and control of one or more of the glands of internal secretion, and iron content, both in erythrocytes and blood plasma, cannot be an exception to this rule. This so important a function cannot be left to chance. We see a higher than normal plasma iron content in certain anæmias and a lower than normal in other types, and this variation is more or less fixed for each type of anæmia.

Treatment so far has been startlingly successful, though the number of cases and the duration under observation, are both inadequate to permit a formal statement. So far, however, large doses of ferrous carbonate have given most satisfying results, not only in improving the anæmia but in relieving many of the secondary distressing symptoms of pregnancy. These, heretofore, have been attributed to the pregnancy, but are most certainly secondary consequences of the anæmia. Further studies are in progress to confirm these, and to place the conclusions beyond any possible doubt. These will be embodied in a later report.

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PRESACRAL SYMPATHECTOMY AS A TREATMENT OF OBSTINATE DYSMENORRHŒA*

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SINCE Jaboulay[1] in 1898 first attempted to relieve pelvic neuralgia by interrupting the different pathways in the sacral sympathetic chains a great deal of work has been done with varying results. His operations were quite complicated and did not give lasting relief to the patients. His work therefore failed to hold the attention of other surgeons at the time and his procedures were quickly forgotten. In 1925, Leriche[2] made a complete study of the pelvic sympathetic nerves in relation to pelvic neuralgia, and introduced the peri-arterial sympathectomy of the internal iliac artery as a means of relieving the pain in cases of functional dysmenorrhœa. This procedure was widely accepted in Europe, and excellent results were soon published by Leriche and Cotte and other foreign workers.

In 1925, Cotte[3] discovered that a simpler operation, such as resection of the superior hypogastric plexus, or neurectomy (of the presacral nerve of Latarjet[4]) also gave complete relief in cases of functional dysmenorrhœa. He suggested that this type of neurectomy be done for pelvic pain and discomfort such as is associated with inoperable cancer of the uterus. For such unfortunates this operation is a boon, and has rendered their existence in this world more tolerable. In cases of functional dysmenorrhœa it should be done only after the ordinary medical measures have been tried, since the operation naturally carries with it at least the risk of a laparotomy.

In November, 1931, Cotte[5] published an account of the remote results observed in 36 patients whom he operated upon during the years 1924 and 1925. In one group consisting of 22 patients no pathological conditions of any kind had been found to account for pelvic pain. In the remaining 14 there were lesions of the genital apparatus which had demanded some intervention upon the ovaries or tubes at the time this nerve was resected. Cotte was able to follow all these cases up and found that after six years all were relieved of the dysmenorrhœa with the exception of one. There were three pregnancies, all the patients going to full term and being delivered without any mishaps. The one woman who had not been fully relieved of pain claimed that she suffered much less, and as he had not been able to make a personal examination of her present condition, Cotte was

* Read before the Mount Sinai Clinical Society on November 21, 1935.

inclined to believe that other factors, perhaps unrelated to the nerve section or the original cause of dysmenorrhœa, might have entered in. Other foreign investigators have duplicated his excellent results.

Behney,[6, 16] Greenhill and Schmitz,[7] were among the first to perform this operation in the United States, to relieve pain in pelvic malignancy. The former has since done presacral sympathectomy for obstinate dysmenorrhœa and reports excellent results. Splendid results in dysmenorrhœa have also been reported by Adson and Masson,[8] Counsellor and Craig,[9] De Courcy,[10] and Wetherell,[11] the last reporting a case of a woman who subsequently became pregnant and was delivered of a live baby, with no untoward changes in pregnancy or delivery. Recently Keene[12] and Pemberton[13] have also contributed some work on this subject with very good results, so that presacral sympathectomy is now accounted a valuable addition to our therapeutic armamentarium, because it can be counted upon for relief of dysmenorrhœa when other methods have failed. A satisfactory result must include unimpaired function of the bladder and rectum, because this operation severs the chief nerve supply to these organs as well as the uterus.

The purpose of this paper is to review briefly the anatomy and technique involved in this operation and to report the results of presacral sympathectomy in three cases of obstinate dysmenorrhœa and also its affect on pregnancy and labour in one case. The patients were operated on by Dr. A. I. Willinsky and myself.

Anatomical Relations of the Presacral Nerve

The presacral nerve lies in what is called by Elaut "the interiliac space". This is triangular in shape, with the bifurcation of the aorta as its tip, the common iliac arteries, its lateral boundaries, and its base, a line across the promontory of the sacrum. On the left side of this space is the inferior mesenteric artery coursing through the base of the mesocolon. Ordinarily this can be displaced to the left, but the root of the mesocolon may extend across the median line of the triangle, making access to the nerve not only difficult but also hazardous. Covering the interiliac space is the posterior parietal peritoneum through which, in thin subjects, the nerves may be seen. Immediately beneath the peritoneum, or separated from it by more or less fat, is a sheet of fibrous tissue, in which lies the presacral nerve, usually in the form of a plexus rather than of a single nerve. The plexus in its sheath crosses but is easily separated from the left common iliac vein, which is seen as a broad band filling in the upper portion and left side of the triangle. As the nerve approaches the base of the triangle it lies on the median sacral vessels and the prevertebral fascia covering the last intervertebral disc and the fifth lumbar vertebra. A few filaments from the superior hypogastric plexus or presacral nerve course along the common iliac artery.

The inferior hypogastric nerves that are a continuation of the principal bundles of fibres from the superior hypogastric plexus course obliquely from above downward in the lateral rectal space; they then follow the internal iliac artery and give off branches with accompanying branches of these arteries, while a group of fibres from the inferor hypogastric nerve terminate in the musculature of the urogenital trigone, bladder and rectum. A secondary plexus forms on the anterior surface of the rectum, just posterior to the uterus and vagina, and becomes intimately connected to the utero-sacral ligaments.

Fontaine and Herrmann,[14] in a review of the sympathetic innervation of the uterus, tubes and ovary, state that the nerve fibres in the wall of the uterus are derived from the great plexus of Frankenhaüser,[15] which is situated on each side of the body of the uterus and the broad ligament. These plexuses are made up of fibres from both the hypogastric and sacral plexuses. The nerve fibres in the wall of the uterus are distributed mainly in the uterine musculature. The Fallopian tube is supplied by unmyelinated and myelinated fibres derived from the ovarian plexus. The ovary derives its nerve supply mainly from the ovarian plexus, which arises from intermesenteric and renal plexuses and follows the ovarian artery throughout its entire course.

Fontaine and Herrmann believed that the hypogastric plexuses carry the important pathways of sensation from the internal genital organs to the medullary centre, and that resection of the superior hypogastric plexus above the hypogastric ganglion is a safe, simple and effective way of interrupting these pathways in the treatment of functional dysmenorrhœa, as well as a method of treating other forms of severe pelvic pain. Adson and Masson say that presacral fibres undoubtedly carry afferent sensations of pain, but it is their opinion that the relief of pain in functional dysmenorrhœa is obtained not only by cutting the pain fibres but from interruption of efferent fibres in the presacral group, which supply the blood vessels in the genitalia and the musculature of the uterus. According to Fontaine and Herrmann the hypogastric plexus exerts a vasoconstrictor action on the vessels of the internal genital organs, whereas the parasympathetic nerves exert a vasodilator effect.

Technique of the Operation

Spinal anæsthesia helps materially in this operation. Through a midline incision extending a little to the left and two inches above the umbilicus the field of operation is exposed by packing the small intestines upward and to the right and retracting the sigmoid to the left. The landmarks thus exposed are the bifurcation of the aorta, the common iliac arteries, and the promontory of the sacrum. On the left side of this space is the inferior mesenteric artery coursing through the base of the mesocolon. The root of the mesocolon may extend across the interiliac space, overlying the presacral nerve. Under these circumstances, the peritoneal incision is made well to the right of the midline and exposure of the nerve beneath the thick, vascular mass is a difficult procedure. Ordinarily, the posterior parietal peritoneum is

incised in the midline just above the sacral promontory, and the incision is continued upward to the bifurcation of the aorta and downward over the promontory. The flaps of peritoneum are elevated by blunt dissection as far as the common iliac arteries, exposing a sheet of fibrous tissue covered by more or less fat. This sheet of tissue contains the fibres of the presacral nerve. In thin subjects, the fibres can be seen and felt, but a thick layer of subperitoneal fat complicates the situation, making recognition and excision of the nerve more difficult. The separation of the sheath and nerve is made *en masse*, beginning over the right common iliac artery near the bifurcation of the aorta. By gentle blunt dissection the separation is continued over the left iliac vein to the left common iliac artery. As Cotte states, "The tissue surrounding the nerves in this area is very delicate, and its tensile strength is supplied by the nerve fibres which it contains". The sheet of tissue thus liberated is elevated and the dissection is carried downward, avoiding the median sacral vessels, until the division into the hypogastric nerves is recognized. The upper and lower attachments are severed after ligation with fine, plain catgut, to avoid troublesome oozing from the vasa nervorum, and the incision in the posterior parietal peritoneum is closed with a continuous suture of fine catgut.

The reports of the three cases follow. Although an unsatisfactory result was obtained in one case, yet the excellent results in the other two, makes us feel that resection of the hypogastric plexus, in selected cases, is a beneficial operation which will in time find a definite place in the gynæcologist's armamentarium.

CASE 1

Mrs. A.C., aged 24, married. Menstruation began at the age of 14, always regular; slight dysmenorrhœa. Six years ago following a mastoidectomy, while still in the hospital, she developed very severe pains in the right lower abdomen. A diagnosis of acute appendicitis was made by her attending physician, and an appendectomy was performed. However two weeks before her next menstrual period (while still in hospital) she developed similar pains and had no relief until she menstruated. She continued to suffer periodically and consulted another doctor who diagnosed adhesions and operated on her, but without relief from her pains. For two years she was treated medically, including endocrine therapy, with poor results. I first saw her on November 1, 1934, and diagnosed severe dysmenorrhœa of the primary or spasmodic type (a type of dysmenorrhœa uncomplicated by gross pelvic disease). Her other complaints were dyspareunia and obstinate constipation.

On November 17, 1934, a presacral sympathectomy was done and two small subserous fibroids were resected. Six weeks after operation she menstruated without any pain or discomfort and her period lasted about 4 days. Four weeks later she had a normal period without dysmenorrhœa. Dyspareunia had disappeared. She missed her next period and on examination at my office I diagnosed early pregnancy at about 4 weeks. Her pregnancy was uneventful, with the exception of staining and some abdominal cramps on three occasions, which cleared up with a little rest in bed and some analgesics. On October 12, 1935, she was delivered of a normal baby girl. The first stage of labour lasted about ten hours and the second stage approximately one hour. Both mother and baby are in splendid condition.

CASE 2

Mrs. K.M., aged 20, married two years, had had severe dysmenorrhœa for four years and dyspareunia for two years, with chronic constipation. This patient had severe abdominal and lower back pains for two weeks prior to menstruation. This partially disabled her during this time; she had to stay in bed throughout her menstrual period, which lasted three days, and morphia was necessary to make her comfortable. During these four years she had been to several physicians and had had various medications, but no operation of any kind was done.

A presacral sympathectomy was done on May 29, 1935. The patient made an uneventful recovery. The first period following operation was on July 5, 1935. She experienced no pre-menstrual or menstual pains with the exception of two hours' discomfort in the back, but no abdominal pain. The last three periods were absolutely painless. Dyspareunia is no longer present and her constipation is very markedly improved.

CASE 3

Mrs. W.P., aged 36, married, complained of dysmenorrhœa and chronic constipation for 23 years. She was operated upon when she was 19 years of age for this complaint, and a left oöphorectomy was done but with no relief. At the age of 24 an appendectomy was performed, but still without relief. At the age of 31 she had a dilatation and curettage with some relief. However her dysmenorrhœa returned, and when I saw her on April 13, 1935, I suggested a presacral sympathectomy. She was admitted to the hospital on June 4, 1935, and the operation was performed on June 5, 1935, in the usual manner.

This patient has experienced similar dysmenorrhœa as before operation for the last three periods. She claims she has no relief at all, but reports definite improvement in her constipation.

CONCLUSIONS

1. Presacral neurectomy relieves dysmenorrhœa of the essential type in a great percentage of cases.

2. It has no harmful effect on pregnancy and labour.

3. It has no permanent deleterious effects on the bladder or rectum; rather very favourable results in relieving constipation.

4. It should be done only in properly selected cases and only after all other treatments are exhausted.

5. It is also used as a relief measure for excruciating pain in pelvic malignancy.

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SOME EXPERIENCES WITH CYCLOPROPANE AS AN ANÆSTHETIC, WITH SPECIAL REFERENCE TO THE DIABETIC PATIENT*

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THE anæsthetic property of cyclopropane was discovered by Lucas and Henderson[1] at the University of Toronto, and their first laboratory report appeared in 1929. In their study they found very few accompanying untoward effects during surgical anæsthesia with cyclopropane, and regarded this gas as very promising and worthy of clinical trial. This was followed by further laboratory investigation at the University of Wisconsin, and later cyclopropane was used to produce surgical anæsthesia in human beings at the State of Wisconsin General Hospital. In 1934 a preliminary clinical report of 447 administrations for major surgical procedures, incorporating an analysis of post-operative complications in the first 200 cases, was presented by Stiles, Neff, Rovenstine and Waters.[2] In this analysis cyclopropane compared favourably with ether in regard to post-anæsthetic and post-operative sequelæ, and seemed to merit more extensive clinical and laboratory study, of at least one year's duration, before its acceptance or rejection as an anæsthetic agent.

A satisfactory technique in administering an anæsthetic requires time for its acquirement. Later, as the technique is improved upon the results of anæsthetization should also be more favourable, so that subsequent reports might prove to be even more encouraging. The physical and chemical properties of cyclopropane have, in some detail, been considered in previous reports.[2, 3] The concentrations of cyclopropane necessary to produce surgical anæsthesia also are now fairly well established. At the present time it appears that the problem of the diluent gas or gases used with cyclopropane deserves much consideration and study. Inasmuch as cyclopropane is capable of producing anæsthesia in proportions of 4 per cent and upwards it is evident that many possibilities of its dilution exist. Oxygen, we believe, should be kept above 20 per cent, or its approximate percentage in atmospheric air, and this we have always done. However we have seen fit to vary the remainder of the mixture greatly, using either nitrous oxide or nitrogen as the diluent. In prolonged operations, unless the pulmonary ventilation is radically interfered with, as in thoracic surgery, patients seem less likely to be shocked when the oxygen concentration during anæsthesia is held around atmospheric proportions than when double or triple the proportions are used. This is especially noticeable in cyclopropane anæsthesia for central nervous system surgery requiring three and four hours of continuous administration.

The ideal general anæsthetic would be one that is non-toxic, pleasant to inhale, rapid in producing anæsthesia profound enough to yield sufficient relaxation for the surgeon to operate with minimum trauma to the patient; it should be non-inflammable and non-explosive in all concentrations; elimination should be rapid and

* Read before the Pacific Coast Association of Anæsthetists on October 31, 1935, at San Francisco.

recovery free from complications. Nitrous oxide perhaps approaches more closely this ideal than any inhalation anæsthetic agent in use at the present time. Its pleasant, rapid induction, and with recovery with freedom from toxicity and minimum post-anæsthetic complications, together with non-inflammability and non-explosiveness have made every anæsthetist feel that nitrous oxide would be the ideal agent if it were only more potent. Unless, however, nitrous oxide be administered in such high concentrations as to preclude adequate oxygenation or be given with some other more potent agent, such as ether or chloroform, it is usually impossible to obtain satisfactory muscular relaxation for abdominal surgery, even though the patient has been well pre-medicated prior to the induction of anæsthesia.

That prolonged periods of oxygen lack are capable of producing considerable tissue damage is now well established. Ether and, to an even greater extent, chloroform have the undesirable feature of upsetting body metabolism rather severely. The fact that ethylene produced a somewhat more profound degree of anæsthesia than nitrous oxide, in the presence of a higher percentage of oxygen, led to the use of it, or the desire to use it, by an increasing number of anæsthetists. Cyclopropane, which will produce more profound anæsthesia than ethylene without ever an excuse for oxygen want, has been gaining favour for this reason, perhaps above all others. However, until a more complete study, such as has been started at the University of Wisconsin, into the electrocardiographic changes produced during anæsthesia with all agents in common use is at hand, it is probably better to use the less potent agents like nitrous oxide and ethylene where they are capable of producing anæsthesia independent of oxygen lack, and reserve cyclopropane for the cases where a more profound narcosis is required. In the occasional case where an extreme depth of anæsthesia is necessary it is perhaps better to supplement cyclopropane with ether until it can be proved that such a mixture is not less toxic to the heart than an extremely high concentration of cyclopropane.

BLOOD CHEMISTRY AND BLOOD GAS STUDIES IN THE NON-DIABETIC AND THE DIABETIC PATIENT

Examination of the blood for sugar, non-protein nitrogen, and the carbon dioxide combining power of the plasma in 25 non-diabetic patients, made immediately before and following cyclopropane anæsthesia for abdominal surgery, where the anæsthetic risk ranged between fair and poor, and the depth of anæsthesia varied from light to a profound third stage, showed remarkably little change. There was a slight rise in the blood sugar in all cases, varying from 8 to 30 mg. per cent above the pre-anæsthetic level. The non-protein nitrogen was influenced but slightly. The occasional changes of from 2 to 5 mg. per cent higher or lower than the pre-anæsthetic level were within the bounds of experimental error. The carbon dioxide combining power of the plasma showed a rather slight but constant downward trend ranging between 2 and 7 volumes per cent. In general, one could say, the blood chemistry and the carbon dioxide combining power of the plasma deviated very little from the pre-anæsthetic level during prolonged and profound anæsthesia with cyclopropane in the non-diabetic patient.

In 30 cases of cyclopropane anæsthesia for various intra- and extra-abdominal operations on controlled diabetic patients blood sugar determinations and carbon dioxide combining power of the plasma analyses were made. The average rise in the blood sugar of only 7 mg. per cent and the slight decrease in the carbon dioxide combining power of the plasma of less than two volumes per cent suggest the value of

TABLE

Agent	*Blood sugar milligrammes per cent*	*Carbon dioxide combining power of the plasma*	*Duration of anæsthesia*	*Number of cases*
Nitrous oxide	Pre-op. 196.0	vol. per cent 56.4	17 min.	30
	Post-op. 200.0	52.7		
Procaine by subarachnoid block	Pre-op. 194.4	54.7	31 min.	30
	Post-op. 195.1	51.9		
Cyclopropane	Pre-op. 175.0	54.9	31 min.	30
	Post-op. 181.9	53.2		

This table showing relative values for blood sugar and the carbon dioxide combining power of the plasma three hours before and four hours subsequently to anæsthesia with nitrous oxide, procaine by subarachnoid block, and cyclopropane, in diabetic patients. The calculated dosage of glucose and insulin was given pre-operatively after the blood sample was taken. No glucose or insulin was administered post-operatively until the second blood sample was taken.

cyclopropane anæsthesia in surgery on the diabetic patient. Further comparisons of cyclopropane and the other agents commonly used to produce anæsthesia in diabetic subjects, namely, nitrous oxide by inhalation and procaine by subarachnoid block, are presented here in tabular form. It appears that cyclopropane compares favourably with nitrous oxide and is superior to sp. subarachnoid block for the production of anæsthesia in the diabetic individual. Bourne[4, 5] has demonstrated that cyclopropane does not damage the liver. This observation is rather important when the part which the liver plays in the metabolism of sugar is considered, and may in a measure explain the remarkably few disturbances of metabolism noted during cyclopropane anæsthesia in the diabetic patient.

Since working with cyclopropane intensely with the Wisconsin group, we were without a supply of the gas at the Philadelphia General Hospital for a period of two months, and during this time we reverted to the use of the older agents. This afforded an opportunity to evaluate clinical impressions gained while using cyclopropane and perhaps better estimate its usefulness. Certain changes in the methods of preliminary medication which have been helpful to us in producing a smooth cyclopropane anæsthesia will be described, and original ideas which we still believe to be essential will again be emphasized.

Preliminary Medication

Optimum preliminary medication, which very materially aids in the subsequent production of anæsthesia with other agents, is a most desirable adjuvant to cyclopropane anæsthesia. The psychic sedation produced by scopolamine, together with the somatic sedation of morphine, have proved to be a very satisfactory combination and the one most frequently prescribed by us, although others have used the barbiturates or basal doses of tribromethanol for this purpose. In view of the anæsthetic potency of cyclopropane, doses of opium derivatives, tribromethanol, or barbiturates large enough to produce muscular relaxation are unnecessary, and because of the decreased pulmonary ventilation caused by them, hinder the speed of the induction of anæsthesia with cyclopropane which has no respiratory stimulating properties of its own. The dosage of opium derivatives has, therefore, been reduced to a fraction of the amount ordinarily prescribed when other gases are used for anæsthesia. For example, in a case for which morphine sulphate, one-fourth of a grain, and scopolamine, one one-hundredth of a grain, would be the selected medication prior to nitrous oxide or ethylene anæsthesia, if cyclopropane was substituted, the dosage of morphine should be reduced to one-sixth or one-eighth of a grain, while the dose of scopolamine should remain unchanged at one one-hundredth. The length of time prior to anæsthetization that the preliminary medication is administered is as important as the dosage of the drug or drugs given to produce psychic and somatic sedation. The maximum effect should be obtained at the time the mask is applied to the patient's face, and with the morphine-scopolamine combination this occurs in the average person about one and one-half hours after its hypodermic administration, and not within twenty minutes or one-half hour as is so generally assumed. Routine orders for preliminary medication are mentioned only to be condemned. Each patient should be seen by the anæsthetist and prescribed for individually, taking into consideration such factors as age, sex, vigour, apprehension, pain, the presence or absence of fever, drug addiction, and others which influence body metabolism. An ideally pre-medicated patient enters the operating theatre with a carefree and slightly euphoric attitude.

The Technique of Administration

The carbon dioxide absorption method was used in all cases. The two variations, namely, the Waters' "To and fro" absorber[6] and the circuit filter[7] will be described as one technique, although from the standpoint of efficiency, conservation of body heat, and lack of resistance to respiration, the Waters' method is probably the better.

Induction

We favour the slow induction of cyclopropane anæsthesia, with the patient breathing through soda lime from the very beginning. The method is a very pleasant one and the subjective sensations experienced by the patient are fairly constant. In a typical case, immediately after the mask is applied and the gas allowed to flow, there is at first a feeling of confidence and surprise when the expansive sensations and tinnitus ordinarily associated with the induction

stage of anæsthesia do not appear. Then, as the concentration of cyclopropane becomes greater, there is a feeling of warmth, quickly followed by a sensation of levitation, and one seemingly floats out into oblivion, a truly wonderful experience. So constant is this sequence of events that the anæsthetist can accurately describe to the patient the sensations which he is about to experience and this materially aids in increasing his confidence. To induce anæsthesia the breathing bag is filled, but not distended, with oxygen and the mask is applied to the patient's face. Then he is allowed to breathe several times into this enlarged respiratory area where the carbon dioxide is removed by soda lime before any cyclopropane is added. This makes possible the detection of any leaks in the system prior to the introduction of the gas. With a leak proof set-up established, cyclopropane is allowed to flow at the rate of from 300 to 500 c.c. per minute for a period of from three to eight minutes, depending upon the size of the breathing bag and the patient to whom the gas is being administered. Oxygen is supplied constantly to meet the metabolic requirements of the patient. When the desired plane of anæsthesia is approached the administration of cyclopropane is discontinued and its effects noted for two or three minutes before further increasing the concentration. This will result in an induction slower than that produced by nitrous oxide or ethylene, but much more rapid than by chloroform or ether. Usually mid-third-stage surgical anæsthesia will occur within six to eight minutes. Should more profound anæsthesia be required additional cyclopropane may be added at the rate of flow of 300 to 600 c.c. per minute, while the anæsthetist watches closely the signs of progressively deepening anæsthesia. The plane of anæsthesia at any given time can readily be ascertained by observation of respiration and reflex activity,[8] which, with a few exceptions, parallels the phenomena of ether anæsthesia. Irregularities of the pulse, which are usually associated with a progressive slowing of its rate, are sometimes seen during mid-third-stage anæsthesia with cyclopropane. This has, in our experience, occurred when the induction has been hastened either by an excessive carbon dioxide stimulation or by giving an unduly high concentration of cyclopropane, or by a combination of these two errors. The remedy is to lighten the anæsthesia by diluting the mixture with pure oxygen. Should more profound anæsthesia be required to produce muscular relaxation in a resistant individual it is preferable to add ether to the cyclopropane rather than push the cyclopropane alone to toxic proportions.

MAINTENANCE

To maintain a constant plane of anæsthesia during prolonged administration, even after the tissues have become saturated, it may be necessary to add small amounts of cyclopropane to replace the gas which is lost by diffusion through the breathing bag and possibly through the patient's skin. With a leak-proof set-up, such as can be obtained by means of an endotracheal catheter surrounded by a Waters' inflatable cuff, it has been possible to maintain anæsthesia for one and two hours after the anæsthetic level desired had been reached without the addition of more cyclopropane. This has been well demonstrated during the course of administration for central nervous system surgery. Difficulty is sometimes encountered in producing the relaxation necessary in upper abdominal surgery, and in such cases, as aforementioned, small supplementary amounts of ether are used. In order to lighten the anæsthesia several methods have been tried; first of all diluting the mixture with pure oxygen, secondly, diluting with nitrous oxide, and, thirdly, and very recently, upon the suggestion of Dr. Ralph Waters, diluting with air. The last seems more reasonable since the pulmonary alveoli are left distended with the inert and slowly absorbed nitrogen rather than the diffusible nitrous oxide. If the oxygen concentration during anæsthesia has been much above atmospheric proportions the gradual reduction of its partial pressure will be beneficial to the patient.

RECOVERY

Most patients regain consciousness within six or eight minutes and the occasional one enters a stage of delirium, which is usually of short duration, several minutes prior to this. After consciousness has returned it is quite common to note patients again passing into a slumber from which they can readily be aroused. Upon sudden removal of the mask, where the oxygen concentration was very high, some patients develop a greyish-blue cyanosis, a marked fall in blood pressure and a bradycardia; this condi-

tion is suggestive of a severe vaso-motor depression, precipitated by a sudden withdrawal of an atmosphere rich in oxygen, humid and warm. The condition is remedied by oxygen and carbon dioxide and is prevented by reducing the oxygen concentration slowly towards the close of cyclopropane anæsthesia.

After Effects

Nausea and vomiting, which are encountered more frequently with cyclopropane than with ethylene, can be reduced appreciably by giving Bourne's buffer phosphate solution immediately following anæsthesia. This clinical observation, which is very striking, has scientific support in the finding of Waters and Schmidt[3] that a marked increase in the blood phosphorus with a quick return to normal occurs during cyclopropane anæsthesia. Stehle and Bourne[9, 10] showed that in animals and human beings there is a marked increase in the phosphoric acid excretion during the anæsthetic and post-anæsthetic periods of ether, chloroform, and nitrous oxide anæsthesia. The primary acidosis of anæsthesia was ascribed by them to the discharge of phosphoric acid from the muscles which neutralizes part of the blood alkali. Bourne[11] suggested giving a buffer phosphate solution consisting of:

Potassium bicarbonate ($KHCO_3$), gram. 100; disodium phosphate crystalline (Na_2HPO_4), gram. 358; distilled water, q.s., litres 2, as a stock solution. The solution used is made by taking one ounce of the stock solution, adding one pint of water and heating to 100° F. This diluted solution is given on basis one pint for each fifty pounds of body weight.

The solution aids in the restoration of phosphoric acid, increases the blood volume, and by reason of its potassium ion stimulates the tissues depressed by narcosis. Potassium acts best in an alkaline solution and especially when the anion is phosphate.

Distention of the abdomen is apparently less frequent after cyclopropane than following ether anæsthesia. During clinical anæsthesia, where the abdomen is open, the intestines are generally observed to be somewhat contracted and active, which contrasts with the ballooned-out, paralyzed gut of ether anæsthesia. Leake and Peoples[12] state that cyclopropane in concentrations up to 25 per cent definitely stimulates the isolated gut *in vitro* while ether causes its paralysis.

Salivation.—It has been noted that some patients, especially those who received no preliminary medication, exhibited a decided increase in the production of saliva during the induction stage over the amount ordinarily seen under anæsthesia with other gases. If the mouth and throat were cleared by suction no further difficulty was encountered until the patient was almost awake, when the saliva would again well up in the mouth. Robbins[13] observed in dogs that cyclopropane in concentrations ranging between 25 and 40 per cent produced a decided increase in salivary secretions up to 250 per cent during the induction stage, stopped secretion entirely during anæsthesia, and caused another increase up to 350 per cent during recovery. Cocainization of the upper respiratory and buccal mucous membrane or the insertion of an endotracheal tube prior to the administration of cyclopropane prevented any increase either during the stage of induction or recovery, although secretion was suppressed during the anæsthetic period.

Conclusions

1. Cyclopropane is a desirable addition to the anæsthetist's armamentarium but should not replace the less potent gases, nitrous oxide and ethylene, where the latter are capable of producing the required depth of anæsthesia independently of oxygen deficiency.

2. Cyclopropane produces very little demonstrable change in body metabolism in either the non-diabetic or the controlled diabetic patient, in so far as has been determined by the tests mentioned.

3. Bourne's buffer phosphate solution is of definite value in reducing the incidence of post-anæsthetic nausea and vomiting with cyclopropane.

The authors wish to express their appreciation for the excellent cooperation of the Metabolic Laboratories of the Philadelphia General Hospital in making the blood studies on the diabetic patients.

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EMBOLISM IN PERIPHERAL ARTERIES

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IN the past five years the records of the Toronto General Hospital contain 126 cases of embolism, making about 25 per year, or one each fortnight over that period. The emboli were found in the following vessels: pulmonary in 56 cases; cerebral in 35; central artery of retina in 3; peripheral arteries in 30; coronary arteries in 2. Also, Belt,[1] in 1934, reported the post-mortem findings of the Hospital relating to this disease. In 10 per cent of all post-mortems there were pulmonary emboli sufficient to have caused the fatal issue, and the incidence of smaller emboli in conditions usually considered to be common complications of operations, such as pneumonia, collapse of lung, heart failure, etc., was very great, probably about 20 per cent.[2] According to the statistics of the Hospital, embolism in peripheral arteries is being diagnosed more frequently, as is shown in the following Table:

TABLE I

1929	2
1930	3
1931	6
1932	3
1933	6
1934	10

With excellent cooperation between internist and surgeon, the disease is assuming a prominent place in emergency surgery. Next to internal and external hæmorrhage, embolism of the peripheral arteries is one of the most urgent surgical emergencies. Acute appendicitis, intestinal obstruction, perforated viscus, etc., while better treated at the first possible moment, usually will not be followed by the disastrous results from waiting six to eight hours that may be expected from neglect of an embolus for the same length of time.

The occurrence of embolism in the arteries has been noted and commented upon for centuries, but John Hunter in 1768, at St. George's Hospital, was one of the first to suggest the possible good results to be obtained by removal of the embolus. Ssabanejew in 1895 was the first to do an embolectomy, but it proved unsuccessful, as the leg became gangrenous. The first successful operation was performed by Mosay and Dumont[3] in 1911, when they removed an embolus from the femoral artery six hours after the beginning of symptoms. In 1921, Einer Key[4] reported a series of successful cases, and it was following this that this branch of surgery began to grow. In 1928, Petitpierre[5] reviewed the literature and collected 118 cases. In August, 1933, Max Danzis,[6] of Newark, N.J., reviewed the literature from June, 1922, to June, 1932, and found 129 cases.

Most peripheral emboli occur in cases where there is a disease of the heart and blood vessels. In our series of 30 cases the origin of the embolus was as follows, and these figures agree very closely with figures given by Zierold,[7] of Minneapolis, and Danzis,[6] of Newark.

TABLE II

Auricular fibrillation	18
Mitral stenosis	2
Rheumatic myocarditis	2
Coronary thrombosis	1
Subacute bacterial endocarditis	1
Carcinoma of the œsophagus	1
Maxillary sinusitis	1
Breast abscess	1
Myocarditis	1
Abscess of jaw	1
No other disease demonstrated	1

The accident may happen in the acute, subacute or chronic stage, but occurs most frequently during an acute exacerbation of a chronic disease. It seldom occurs as a post-operative complication, unless there is some history of cardiovascular disease. Mitral disease, with or without fibrillation, is the commonest cause of intracardiac thrombus formation on the left side of the heart. Occasionally, embolus may come from thrombus in the right heart, or from phlebitis of peripheral veins, but in these, there must be a patent foramen ovale, allowing the paradoxical embolus to occur.

Welch[8] and Fahraeus and Lindqvist,[9] showed that three factors are involved in development of a thrombus at any site: (1) change in the intima; (2) physiochemical change in blood;

(3) local changes in rate of flow. Changes in the heart cavities or valves due to disease, or changes in force or rhythm of the beat, may change the local rate of flow, and, associated with these changes, the endocardium is usually damaged, thus providing two of the necessary factors for clotting. Occasionally physiochemical changes in the blood play a part. Under these conditions, platelets are precipitated and agglutinated at some point on the endothelial surface. On the damaged intima, in the presence of platelets, fibrin may be formed, entangling the red and white cells, and the mass may grow with considerable speed.

So long as the conditions under which the thrombus formed remain unchanged everything goes on merrily. If auricular fibrillation was the chief factor, by causing a change in rate of flow, nothing unusual may happen as long as the fibrillation continues, but if it changes to a regular and normal beat, either spontaneously or due to medication, immediately the blood flow is changed and a part or the whole thrombus may be dislodged from an auricle or ventricle, or a vegetation from a valve may become detached and pass into the left ventricle, and through the aorta, to become lodged in a peripheral artery. The onset of an attack of fibrillation may dislodge a thrombus. Sometimes there is gross change in heart function to account for the sudden throwing-out of an embolus. In coronary thrombosis a mural thrombus may increase in size until a portion which is less firmly attached may become detached without any obvious exciting factor.

The change in rate of blood flow and possibly physiochemical changes in blood that occur in diseases of peripheral blood vessels and general systemic diseases, such as pneumonia, diabetes, alcoholism, pregnancy, syphilis, tuberculosis, thyrotoxicosis, streptococcal infection, eclampsia, etc., may account for thrombus-formation in the heart and blood vessels. It is possible that many of the unexplained aches, pains, sudden rises of temperature, mild lung complications, etc., that occur after operations are due to small emboli whose source is not discovered, as described by Belt,[2] and where a definite diagnosis cannot be made. The case, however, that attracts our attention for the present is one in which there are definite clinical manifestations.

When a mass of thrombus large enough to produce definite symptoms becomes detached it floats in the blood through the aorta until it enters one of the branches of this artery or reaches its bifurcation. It continues on its course until it reaches a point where the repeated branching of the artery has diminished the lumen so that the solid mass of the thrombus obstructs it. The trunks of the arterial tree for the most part are cylindrical between branches, so that if an embolus passes one branch and enters the next segment of the artery it will not be obstructed until the next branch is given off, at which point the lumen is diminished in proportion to the size of the branch given off. Eventually the embolus is too large to enter the next segment, and so it is stopped at a branch of the artery or at a bifurcation. Emboli tend to lodge at fairly constant points in the arterial tree. The following is a list of these sites in our series of 37 surgical emboli in 30 patients.

TABLE III

Right common femoral	11
Left common femoral	7
Both common femorals	1
Both common iliacs	1
Left common iliac	1
Bifurcation of aorta	4
Popliteal	1
Anterior and posterior tibial	1
Brachial—at superior profunda	3
at bifurcation	1
Axillary, at subscapular branch	2
Radial	1
Mesenteric (superior)	3

An embolus may lodge at a branch or bifurcation and may not obstruct either branch completely, in which case it usually is called a "riding" or "saddle" embolus. It may remain in this position for a varying length of time, then it may slip off into one branch, plugging it completely and leaving the other clear, or a smaller piece may break off and travel in the other branch until it arrives at a point where it also either rides at a bifurcation or plugs one or both branches. If the riding embolus does not plug both arteries it changes the blood flow so that a secondary thrombus begins to form distal to this point, as seen in several cases and as described by Fsähraeus and Lindqvist.[9] It increases in size quite rapidly and may extend from a few cms. to a great distance, as one described by Nyström[10] which was 86 cms. in length. This process does not extend proximally beyond a few millimetres, and in any case not beyond the next proximal branch, provided there is no generalized sepsis. Within a short time this secondary thrombus may complete

the obstruction of the vessel. From that time, the only nourishment available to tissues beyond is through collateral circulation. If the embolus obstructs a vessel completely this secondary thrombus may be non-existent, and in the diagnosis and treatment this point should be taken carefully into consideration.

The breaking-off and escape of the embolus from the heart usually produces no symptoms, but in two of our cases the first complaint was a sensation of precordial distress accompanied by weakness and faintness, followed within a few minutes by peripheral pain. Only the 30 cases with 37 emboli in peripheral arteries will be considered.

SYMPTOMS

Pain.—The first evidence of the lesion in 33 of the 37 cases of embolus was pain, and in the other 4 there was a sudden change in sensation, such as numbness, coldness, or stiffness of the extremity. The pain varied from a moderate aching, indefinitely localized, probably due to an incomplete blocking of important channels by a saddle embolus, to an agonizing pain starting suddenly at a definite spot and extending along the course of the vessels involved to the periphery, and probably due to a sudden complete occlusion of a main trunk. The local pain is probably due to the sudden violent constrictor spasm of the vessel wall resulting from the irritation of the obstructing solid body, and the extension of the pain, to the spasm passing along the vessels to the periphery, leaving them in the contracted state as found at operation. The widespread aching and increasing dull pain is probably due to anoxæmia of tissue, and persists in the absence of sedatives either until gangrene occurs or the circulation is restored. In some of our cases the patients were able to distinguish between the first sensation of pain and the subsequent one extending peripherally, and were able to localize it, as, for example, in the popliteal region, and in some of these, the embolus was found in that region. It may be reasonable to theorize that this occurs only with sudden complete occlusion, as was demonstrated in several patients with this type of pain. In those where there is partial obstruction only these sharply localizing signs are not present, and only a diffuse pain from anoxæmia is present. As the occlusion becomes complete from the secondary thrombus the pain becomes more severe and constant.

There were 4 cases in our group in which probably there was a very small saddle embolus which impaired the circulation, but very slightly, within four hours. However, the secondary thrombus developed to the size where it began to impair and finally to obstruct the blood flow, with development of symptoms of anoxæmia. This latter type of case, beginning with tingling, numbness or coldness, has frequently been classified as thrombosis of a peripheral artery. In our group of cases, there was no evidence either at operation on the affected vessel, in an amputated extremity, or at post-mortem examination, that the lesion was thrombosis. This of course excludes the cases of obvious peripheral vascular disease, such as arteriosclerosis, occurring in the diabetic, or senile patient, Buerger's disease, aneurysm, etc., where there are symptoms of slowly increasing vascular obstruction over a considerable period of time. It is probably true also that the symptoms of sudden, violent, localized pain can be produced only by a vessel whose wall is composed chiefly of muscle. As is quite well known, the smaller arteries toward the periphery have a greater proportion of muscle in their wall, and the larger ones, like the common iliacs and the aorta, have chiefly elastic and fibrous tissue which has no power to go into constrictor spasm. This probably explains the fact that in the cases of embolism of the bifurcation of the aorta and of the common iliac vessels the patients complained of no severe pain until gangrene developed, and the early symptoms were of numbness, tingling, coldness, and stiffness of the feet and legs, with some aching later, indicating more serious obstruction. In these cases there were cyanotic colour changes in the skin, and pallor appeared before severe pain developed.

Colour changes.—In the absence of colour changes in the skin, either in the form of pallor or cyanosis and mottling, it may be assumed there is no lesion of the arterial tree that will endanger the extremity. The changes in the presence of a sudden complete block are usually pallor of the distal parts of the extremity, changing to a band of cyanosis, and proximal to that an area of blotchy mottling, fading into the normal colour. The level of

these colours is of very great importance in helping to decide the level of the obstruction. It is to be remembered that the normal colour of skin extends considerably more distal on the extremity than the level of the block, due to the collateral circulation. If the extremity is dependent, it may not show pallor in the presence of a complete block, and if it is elevated this sign is more marked. The findings here are taken from observations in this group of patients, lying in a horizontal position, and from descriptions in their records. If the upper margin of colour change is at or below the instep the embolus is probably lodged at the bifurcation of the posterior tibial artery, or there is an incomplete obstruction higher in the posterior tibial artery. If this line lies between the ankle and a hand's breadth below the knee joint, the obstruction is probably at the popliteal bifurcation or there is incomplete obstruction more proximally. If the line is above the knee joint and below the upper third of the femur the embolus is obstructing both superficial and profunda femoris arteries, or, in other words, the bifurcation of the common femoral. If the upper third of the femoral region is involved the bifurcation of the common iliac is the site, and if the line extends to the lower abdomen, involving both sides and both legs, it is at the bifurcation of the aorta. At any of these sites, if the obstruction is incomplete, the whole picture is changed. In none of our cases have emboli been found at points other than those mentioned, and in reading the case reports of other writers their findings would appear to coincide.

Temperature changes are present in all cases where there is serious obstruction of main arterial trunks in the extremities. The area showing pallor rapidly becomes cold and drops to room temperature. Proximal to this, over the area of cyanosis the skin is cool, reaching normal temperature only at the level at which skin colour is found to be normal. This sign is also a valuable aid in localizing the level of arterial obstruction.

Sensory changes are inconstant and not very helpful in making a diagnosis. Normal sensation extends far below the level of obstruction, followed by an area where there it patchy anæsthesia, with probably complete anæsthesia only if the lesion involves the larger main arteries nearer the trunk.

Paralysis is variable and of little practical importance in localizing the lesion. The patient frequently can move the toes when they are pallid and anæsthetic.

Pulsations in the peripheral vessels are probably the most important single finding, and, used in conjunction with the colour and temperature changes, usually suffice to localize the lesion. If pulsations are palpable in the posterior tibial artery behind the internal malleolus and in the dorsalis pedis artery, it is safe to assume that there is no embolus in the vascular tree of that extremity that will endanger the foot, and the same applies to the radial and ulnar arteries with relation to the hand. If, however, the arteries of the foot are not pulsating, and the femoral and upper part of the popliteal are pulsating in the presence of other findings, the obstruction is at the bifurcation of the popliteal. Similarly, if the popliteal and foot vessels are not pulsating and the common femoral is pulsating, the lesion is at the bifurcation of the common femoral artery, if there are coldness and colour changes above the knee. If none of the arteries below Poupart's ligament pulsate, the obstruction is at the bifurcation of the common iliac, and if both extremities are involved in this way, with the collateral findings, the bifurcation of the aorta is the site of the obstruction.

In the upper extremity coldness and colour changes of the hand to the wrist and lower third of forearm, with absent pulsation below the level of the condyles of the humerus over the brachial artery, place the obstruction at the bifurcation of the brachial artery. If the middle third of the forearm is reached by these changes, and pulsation is felt only in the upper third of the brachial artery, the embolus is lodged at the origin of the superior profunda from the brachial artery. If the hand and forearm are involved to the elbow region, the origin of the subscapular artery from the axillary is probably the site. There have been no cases of embolus of the subclavian or innominate artery in our series.

The oscillometer is of value in some cases, but showed only diminished pulsation, probably due to collateral circulation, in some of which cases an embolus was found and removed. Arteriography with thorotrast, sodium iodid, etc., was tried experimentally, but is not used, as the U. S. Pharmacopeia has not accepted it

because of the danger involved due to the radium content of the thorium dioxide. The permanent injury to blood vessels is a danger, because of the concentration required and the obstruction preventing flushing of the area by the bloodstream. Perlouis' histamine test for collateral circulation and the intracutaneous saline test were not found to be of much value in this disease. In differential diagnosis the presence of previous peripheral vascular disease with thrombosis and thromboplebitis should be excluded.

TREATMENT

Having made a diagnosis on the history of previous heart disease, onset, and physical findings, and having localized an embolus, the problem of treatment must be considered very carefully. As recently as 1932 Neuhof[11] of New York discouraged surgical treatment on the ground that most cases would be just as well without operation. Analysis of our cases supports the opinion of other writers that there are few operations in surgery so eminently satisfactory in selected cases or attended by such potentiality for good as embolectomy for arterial embolus. The method advocated feebly by Key and Nyström, namely, massage to break up and shift the embolus to more distant vessels where collateral circulation might be sufficient, has not been tried. The contraindications to operation are found in those cases where (1) a positive diagnosis is not possible; (2) where the accident was known to have occurred many hours before, as shown by the results which will be mentioned; (3) and those in which the patient has multiple emboli, probably in cerebral and mesentric vessels as well, or those in which any surgical undertaking is unwise because of poor general condition. It was found that amazingly bad risks, in the form of heart failure, coronary thrombosis, etc., stood the operation very well. This is partly accounted for by the fact that many of the emboli can be removed under local anæsthesia. In the cases of aortic embolism, of which we had three, which were operated upon successfully the patient stood the operation and the general anæsthesia very well.

OPERATION

The affected area is exposed, the vessels distal to the obstruction are found collapsed and in spasm, so that they appear from one-half to one-third their normal diameter, and there is no pulsation. Usually at the bifurcation of a vessel there is a segment showing distension with a bluish colour, which does not pulsate, is firm on palpation, and immediately proximal to this the vessel is normal in colour, size, shape, and pulsates forcibly, both to vision and palpation. The field is kept flooded with 2 per cent sodium citrate. Soft rubber-covered clamps are applied well distal to the area thought to contain secondary thrombi, and also proximal to the thrombus. The adventitia is cleared over a small area and a longitudinal incision, 0.5 to 1 cm. long, is made. The mass is expressed by the fingers without difficulty and the lower clamp is removed to allow return bleeding to flush the distal segment and similarly the proximal segment is flushed and the clamp reapplied. With fine No. 000,000 oiled silk suture on arterial needles, the incision is closed with continuous suture, and the clamps are removed. The area of dissection and the peripheral segment of the vessel remain in spasm, but, in spite of this, if the embolus has been removed successfully pulsation may be felt below the original block, and often in the distal part of the extremity. This spasm seems to relax over a period of many hours, as the pulsation usually improves, reaching its maximum in about 48 hours in our cases. If successful, the colour, temperature, anæsthesia, and paralysis improve promptly. In some cases there is improvement in these findings without a return of pulsation in the superficial vessels. Several cases are reported which have been treated by the Pavex machine, in which pressure can be changed from a negative to a mild positive reading. This treatment aims at establishing a collateral circulation early enough in the disease to prevent gangrene. It is felt, however, that if the main arterial channels can be restored safely and satisfactorily the best possible results will be obtained.

RESULTS

In the 30 cases of peripheral embolism operations were performed on 17, and in 13 there was no operation. Of the 17 patients operated upon, 9, with 11 operations, are alive and well, with adequate circulation to the extremity. Of the 8 remaining, 1 is alive and well, but required amputation of the leg. One patient had good return of circulation for 24 hours, but then

symptoms and signs of gangrene returned, probably from thrombosis at the site at which the embolus was lodged, with a fatal result. Six patients had a satisfactory return of circulation and the extremity was saved, but the patients died several days later with other complications such as, cerebral embolus, pneumonia, pulmonary embolus, etc. This makes 16 cases in which the operation was successful in restoring the circulation. Only one of these surviving cases was successful when the operation was undertaken more than six hours after embolism occurred.

Of the 13 patients with no operation 8 developed gangrene in one or both legs, with a fatal issue in all.

TABLE IV

INTERVAL BETWEEN EMBOLISM AND OPERATION

	Up to 6 hrs.	6-12 hrs.	12-18 hrs.	18-24 hrs.	Over 24 hrs.
Patients alive and well with good circulation	8	0	1	0	0
Number of patients in whom circulation was restored, but died of complications	4	2	0	2	0

In 4 cases where the circulation did not appear to be badly affected, there was improvement without operation, and gangrene did not develop. One embolus was at the bifurcation of the brachial artery, one at the common femoral artery, one at the posterior tibial, and in one other, thought to be embolus of the mesenteric artery, but not proven, recovery took place. In four later cases where gangrene was present on admission to the hospital primary amputation was performed, with death in all.

CONCLUSIONS

1. Embolism in peripheral arteries is not uncommon.

2. The diagnosis can usually be made without difficulty by careful observation of the clinical signs and symptoms.

3. In early cases, embolectomy offers good results.

4. Neglected cases usually terminate with amputation or end fatally.

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A CASE OF RECTOVAGINAL FISTULA AND STRICTURE OF THE RECTUM DUE TO LYMPHOGRANULOMA INGUINALE*

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LYMPHOGRANULOMA inguinale is the so-called "sixth venereal disease" (Stannus) and undoubtedly is responsible for a certain group of strictures of the rectum. It is interesting that textbooks of even recent date list as the cause of strictures, gonorrhœa, tuberculosis, and syphilis; I think there is little doubt that many strictures are due to the virus of lymphogranuloma inguinale. The diagnosis of the disease discovered by Durand, Nicolas, and Favre was definitely made accurate by Frei in 1925. The intradermal test is now known by his name.

Many terms in the past have been used to describe this condition. The "esthiomène" of the old textbook is probably the lymphogranuloma inguinale of today. A descriptive account of the rectal involvement was given by Fournier in 1875. In 1849, Larsen, a Danish surgeon, reported three cases of "hyperplastic infiltration" of the rectum, in one of which, as in that I am reporting, there was a rectovaginal fistula. Syphilis being rampant at that time, Larsen considered his cases due to it.

A number of investigators are of the opinion that an etiological factor in the production of strictures is a diversion of the lymph stream

* Read before the Section of Obstetrics and Gynæcology, Academy of Medicine, Toronto, November 7, 1935.

toward the rectum as a result of destruction of the inguinal glands. The rectal glands now suffer from the same affection and the inflammatory reaction so produced leads to a stricture. These rectal glands, first discovered by Gerota, of the Waldeyer Laboratory, bear his name today. These glands, six to eight in number, receive the lymph from the lower portion of the rectum and are situated directly on the tunica muscularis, between it and the fascia propria, and extend from above the insertion of the levator ani at the rectum to the level at which the peritoneum reaches the lateral walls.

CASE REPORT

Mrs. W., aged 25; born in Canada; married two years; one previous pregnancy.

Shortly after the New Year, 1934, the patient experienced pain in the lower abdomen and noticed a few small lumps in both groins and over the saphenous openings. She also had some rectal distress and thought the circumference of the stool was smaller. In February the abdominal pain became more marked and the swellings larger, more tender, and one or two broke down and discharged a yellowish pus. In the course of two weeks' time she also noticed a purulent, sanguineous discharge from the rectum. At this time she consulted her physician who opened the other inguinal abscesses and applied alcohol compresses. Her condition did not improve. The rectal symptoms grew worse and I saw her with her physician on May 8, 1934.

Examination revealed numerous sinuses in the inguinal and saphenous areas discharging pus. The lower abdomen was tender, and vaginal examination showed pelvic masses around the region of the great vessels. The uterus itself was free. A rectovaginal fistula was present, the opening in the rectum being about 3 inches above the anus. A well marked stricture of the rectum was present about 2½ inches above the anus, and the rectal wall seemed very thick, was extremely tender, and contained three to four definite nodules in the rectovaginal septum. She had lost 20 lbs. in 6 months' time. There was no menstrual disturbance. After an exhaustive enquiry I found that her husband had also suffered from inguinal abscesses with sinuses. A tentative diagnosis of lymphogranuloma inguinale was made and the patient was admitted to the Toronto General Hospital. She was afebrile. Dr. Philip Greey, bacteriologist at the Banting Institute, saw the patient with me and carried out a Frei test on both husband and wife. Both tests were positive, the latter very strongly positive. This settled the diagnosis.

The laboratory findings were as follows: The Wassermann test was negative. Urine (catheter specimen), negative. White blood cells, 8,000; red blood cells, 5,200,000; hæmoglobin, 90 per cent; sedimentation time, 60 minutes. Urethral, vaginal, cervical and rectal smears were negative for gonococci. A swab from the perineum on culture showed a *S. aureus* and *Strept. hæmolyticus.*

A barium enema showed a stricture of the rectum; the descending colon was free. Treatment was commenced, consisting of intravenous injections of a 1 per cent solution of tartar emetic in saline, starting with 2 c.c. and increasing to 7 c.c. twice a week. At the end of 6 weeks' time she had received 75 c.c., had experienced very little reaction from the injections, and the inguinal sinuses were healed. The rectal discharge had improved greatly and the "pelvic neuralgia" had subsided. The patient did not receive any lymphogranuloma inguinale antigen as we had difficulty in securing it. She made such a steady and marked improvement on the tartar emetic that we were glad we had not mixed the treatments.

She was seen last on March 15, 1935. The following notes were taken.

A stricture of the rectum is definitely established, the circumference of the stool is small. There was no blood on the examining finger but the stricture area is very tender. The nodular masses in the rectal wall have disappeared. The pelvic glands are not palpable and the external sinuses have healed. An irregular, tortuous, fistulous tract is present between the vagina and the rectum. The opening in the rectum is at the site of the rectal stricture, and the opening in the vagina is on the posterior vaginal wall, just inside the introitus. I think the tortuosity and length of the fistulous tract can be explained by a breaking down of the numerous glands of Gerota in the rectovaginal septum and rectal wall during the acute phase. The abscess had opened into both rectum and vagina with a resulting fistula. The Frei test is still positive and will remain so for the rest of her life.

I cannot accept the theory prevailing that the rectal stricture is due to a diversion of the lymph stream following an inguinal stenosis. The case just reported would lead me to a different conclusion. May I offer the suggestion that the virus leads to a widespread glandular involvement of the pelvic, anal, inguinal, and rectal glands at the one and same time. At no time was there any elephantiasis of the vulva or anus. The history was that of abdominal pain, swollen inguinal glands and rectal distress. The glands of Gerota apparently broke down at the time that the inguinal glands started to discharge.

SUMMARY

1. A case of rectal stricture with rectovaginal fistula is reported which reacted positively to the Frei test.

2. A suggestion is offered that the affection is a widespread involvement of the inguinal, anal, pelvic and rectal glands, at the one and the same time, and that the inguinal and pelvic glands respond to treatment much more quickly than the rectal, and that treatment of the latter is very stubborn.

3. The treatment instituted caused a rapid healing of the inguinal sinuses and a complete disappearance of the pelvic glands.

4. A plea is made for a Frei test and rectal examination in all cases of inguinal adenitis. Early diagnosis and appropriate treatment is essential to the prevention of a rectal stricture.

5. Lymphogranuloma inguinale is a bad term. It is a more extensive disease than that term would imply, and the rectal involvement is the important and dangerous feature.

I wish to record my thanks to Dr. Philip Greey, bacteriologist at the Banting Institute, for carrying out the Frei tests for me.

A CASE OF GASTRIC ULCER CURED BY GASTRECTOMY AFTER FORTY-FIVE YEARS OF TREATMENT

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THIS case is an interesting example of the type of gastric ulcer, which, in spite of all medical and surgical treatment, progresses from bad to worse. These patients are often finally told that nothing further can be done for them and are thus condemned to a life of pain and chronic invalidism.

This patient had a history of 45 years of symptoms, with persistent treatment and hospitalization, including three gastric operations. Finally a subtotal gastrectomy was decided upon and successfully carried out. This was three years ago (December, 1932) and since that time she has been perfectly free from symptoms. It is the author's belief that a large proportion of such cases can be cured by similar operations, provided certain important technical details (to be mentioned later) are observed.

CASE

Mrs. W.R. entered the Royal Victorial Hospital for the first time in 1914, complaining of recurrent attacks of severe abdominal pain and vomiting. She suffered her first attack of acute abdominal pain and vomiting about 1886, at the age of seventeen. After a week in bed at that time on liquid diet she was relieved of her symptoms for six months. Before coming to Canada she was in the Sheffield Infirmary on six or more occasions, suffering from repeated attacks of epigastric pain and vomiting, interspersed with periods of relief, which, however, never lasted for more than a year. She was never able to eat a regular diet but lived on gruel, tea, jelly, and milk puddings. Her attacks of pain and vomiting lasted about a month to six weeks, each attack increasing in severity. X-ray showed a niche on the lesser curvature of the stomach with an incisura. She was put on a strict ulcer régime and discharged improved. In 1916 on account of very severe vomiting, she was re-admitted, when the diagnosis was confirmed by x-ray. The stools on this occasion showed occult blood. After a period of bed rest and strict diet she was again discharged improved. In the same year the patient was admitted to the surgical side when the ulcer was excised.

Her next admission to the Royal Victoria Hospital was in 1923. The patient had been completely well for a time following her operation, but recurrent attacks of pain and vomiting, occurring every six to nine months necessitated her re-admission. She had been repeatedly in bed under strict ulcer régime. X-ray again showed a niche on the lesser curvature, with an hour-glass stomach. On this admission she underwent a gastro-enterostomy. The following year, 1924, she was again admitted for recurrent attacks of pain and vomiting. Gastro-enterostomy had only succeeded in giving her eight months' relief from symptoms. X-ray showed a functioning gastro-enterostomy stoma draining the lower part of an hour-glass stomach. Bed rest and Sippy diet improved her symptoms and she was discharged on a diet.

Two years later (1926), she was again admitted on account of progressively more severe attacks of pain and vomiting. X-ray showed a recurrent ulcer, in spite of a functioning gastro-enterostomy. Gastric analysis showed hyperacidity. The patient again improved on bed rest and a Sippy diet and was again discharged on a diet.

In 1927 she was again admitted with identical findings, and again discharged on a diet after the relief of her symptoms. In 1928 she was admitted with the same old symptoms and another operation was performed. Stomach resection did not appear feasible on account of the numerous adhesions, so the gastro-enterostomy was undone and the stomach and jejunum restored to their normal relationship. In 1930 she was again admitted to hospital after three increasingly severe attacks of pain and vomiting, the last of which was not relieved by bed rest and a Sippy régime. With intravenous glucose-saline feedings and bed rest, her symptoms gradually subsided and she was again discharged. X-ray on this occasion showed a large ulcer on the lesser curvature with an hour-glass stomach.

A few months later, in 1930, she was again admitted with a return of her old complaints. Pain was progressively severe, and she was unable to keep anything on her stomach. X-ray confirmed previous findings. For twelve months she remained free from symptoms but 1931 found her back in the medical ward, as bad or worse than ever. Examination of the stools showed occult blood. She was fed parenterally until a diet could be tolerated.

On admission the following year (1932) no improvement being evident, her attacks, in fact, being progressively worse, she was seen in consultation by Dr. F. A. C. Scrimger and the author, and subtotal gastric resection advised. She was transferred to Surgery and in December, 1932, was operated on by Dr. Scrimger. Spinal anæsthesia was used. The stomach was imbedded in adhesions. The ulcer, three-quarters of an inch in diameter, on the posterior wall of the stomach at the lesser curvature, penetrated the pancreas deeply. As much of the stomach as could be freed was removed. The ulcer bed was left *in situ*. The jejunum was anastomosed to the stomach anteriorly. A posterior anastomosis was impossible on account of the previous operation with thickening and scarring of the meso-colon. An entero-enterostomy was added on account of the inflammatory condition of the stomach and because the angle at which the jejunum left the stomach was more acute than desired. The patient made an uninterrupted recovery and left the hospital symptom-free.

Three years later in December she was re-admitted for a check-up. She had been completely symptom-free during the interval. She ate and drank anything and enjoyed all her meals. For the first time since 1886 she had no fear of eating. Gastric analysis showed no free hydrochloric acid and a low total acidity. A barium series showed the barium staining the stomach after nine hours and scattered through the small bowel and in the large bowel to the splenic flexure. After twenty-four hours the barium was all in the large bowel. The rate of flow of the barium through the stomach and small bowel appeared to be normal. There was no sign of a recurrent ulcer. There was no occult blood in the stools. There was no anæmia, her red blood count totalling 4,800,000, with hæmoglobin of 92 per cent. For three years she has remained perfectly well and symptom-free.

This history is one of repeated attacks of pain and vomiting. For weeks the patient remained in bed under the strictest medical regime under the care of a competent gastro-enterologist. During her forty-five years of symptoms she was in hospital about twenty times. When she was not in hospital she was undergoing dieting under medical care. Latterly she was only admitted to hospital when the pain and vomiting became unbearable. Since 1914 she had been operated on on three occasions, undergoing an excision of the ulcer (now generally regarded as an almost useless procedure) gastro-enterostomy, (an operation which is rapidly being recognized as quite inadequate), and, finally undoing of the gastro-enterostomy and restoration of the stomach and jejunum to its original state. If a gastro-enterostomy failed to cure an ulcer for which it had been carried out it is difficult to see what benefit could be derived from undoing the gastro-enterostomy. Yet not many years ago this was considered to be the next logical procedure.

The most common characteristic of peptic ulcer before it has penetrated into the pancreas or other adjacent structures is intermittency. After any operation, on almost any dietary regime, temporary relief of symptoms can be expected extending over a period of weeks or months and occasionally years. Even with no treatment this periodicity of symptoms is quite characteristic. This is probably the chief reason why every treatment and so many operations have enjoyed a temporary popularity, only finally to meet a growing volume of criticism. Follow-up results after five years have shown all treatments so far in general use, both medical and surgical, to be inadequate.

Ulcer is an intermittently chronic disease. A review of statistics shows that the average duration of symptoms of patients attending medical clinics is from 5-6 years, and of those coming to operation from 6-10 years. During all these years patients have been suffering symptoms and undergoing treatments at intervals. Lahey states that both physicians and patients accept and continue to accept the original peptic ulcer with all too great complacency. His mortality from hæmorrhage alone while undergoing medical treatment in the hospital is 5 per cent, a greater mortality than any surgeon would like to admit following surgical procedures in uncomplicated cases. Much of the high mortality occurring in gastric ulcer must be laid to the procrastination of the patient and his physician. Once an ulcer has been the seat of a massive hæmorrhage, that hæmorrhage is liable to occur under the strictest medical treatment and even cause the death of the patient. Cases of this type can always be found among the records of any general hospital. Perforation is a frequent possibility and carries with it a hazard of death. Inanition, early carcinomata thought to be and treated as ulcers, penetration of the ulcer into the surrounding viscera, are all possibilities which greatly increase the danger of death. Another great danger is the difficulty of operating a second, third, or even fourth, time, due to preceding unsuccessful and inadequate operations. In a report on gastro-jejunal ulcers, Lahey combed from the literature 1,089, reported since 1928. These ulcers, occurring after gastro-enterostomy, range from 2 to 24 per cent in various reports. In a careful study of 85 patients, Hinton found 16.4 per cent to be suffering from marginal ulcer and believes this an underestimation.

Farquharson in a follow-up study of 480 cases, 200 treated in the medical clinic, 200 treated by gastro-enterostomy, and 80 treated by gastro-duodenostomy, found the following results:

	Medical treatment	*Gastro-duodenostomy*	*Gastro-enterostomy*
A. Completely cured	15.3	28	48.3
B. Improved, with occasional symptoms.....	43.2	44	25.2
C. Improved, but with frequent attacks of pain	32.2	22	21.6
D. Failure	9.3	6	4.9

But 50 per cent of cures following operation is not sufficient, and gastro-enterostomy must be discarded for a more radical procedure.

For many years German and Austrian surgeons have advocated subtotal gastrectomy as the routine procedure for ulcer. Finsterer and v. Haberer, who have done over 5,000 resections between them during the last 15 to 20 years, report a mortality of between 2 and 3 per cent in the uncomplicated cases and 15 per cent in the complicated. Finsterer reports 95 per cent cures after from three to fourteen years. Haberer states his end-results are good, with not over 0.6 to 0.7 per cent being defective. Jean

Duval, following carefully Finsterer's technique, has carried out 108 resections with a mortality of 2 per cent. He has since February, 1934, operated on 53 consecutive cases with no mortality. The author, following Finsterer's technique for gastric resection in every detail, has operated on 23 cases with no mortality. The post-operative course shows no marked discomfort. The follow up results up to the present have been most gratifying.

The development of post-operative anæmia is often used as an argument against resection. This condition does occur, but is practically always of the hypochromic variety, and with suitable treatment clears up. Some of the severest cases follow gastro-enterostomy, and the anæmia appears to be due to the type of operation rather than to the amount of stomach removed. When the food passes through the stomach and small bowel rapidly, gastro-intestinal symptoms occur as well as those of secondary anæmia. The symptoms lead to a dietary regime, frequently inadequate, which is an additional cause of the anæmia (apparently a deficiency anæmia). If the correct technical procedure is carried out these conditions can be avoided in almost all cases.

If a long afferent loop is left proximal to the stoma, food tends to collect in the blind duodenal stump and stagnate there. This will undoubtedly produce gastro-intestinal symptoms.

Gastric resection removes the lesser curvature and the pylorus, the commonest sites for ulcers. A sufficiently radical resection removes a great deal of the acid-bearing mucosa. It has been suggested that resection is illogical because it does not remove all the acid-bearing mucosa. Repeated gastric analysis on resected cases shows a marked lowering or absence of free hydrochloric acid following resection in almost all cases. Further, the stomach empties more rapidly, so that the continued stimulation to acid secretion is lacking and bile tends to regurgitate through the stoma into the stomach, thereby neutralizing the acid. As all the acid-bearing stomach is not removed an entero-enterostomy should never be performed if it can be avoided, as it leads the alkaline secretions of the duodenum away from the stoma, thereby increasing the risk of marginal ulcer.

Reports of results following gastrectomy are somewhat confusing. All reporters have not obtained as good results as Finsterer and Haberer. This, I believe, is due first to the varieties of technique used, and, secondly, to the limited amount of stomach which some surgeons remove. An operation which is little more than a pylorectomy is frequently classified as a gastric resection. Such inadequate procedures will always produce poor results.

In conclusion I would like to urge an unbiased review of this subject by all clinicians, considering as cured no case which has been operated on for less than three years or preferably, five years. The results of Haberer, Finsterer, and Claremont, with their enormous material, should be carefully studied, especially as these men owe their large series to the happy results obtained by their method of treatment.

HISTIDINE HYDROCHLORIDE VERSUS DIET AND ALKALIS IN TREATMENT OF PEPTIC ULCER: IMMEDIATE AND LATE EFFECTS IN FORTY-ONE CASES.—Kirby A. Martin treated forty-one selected patients with acute symptoms and radiological signs of active peptic ulcer with intramuscular injections of histidine hydrochloride (from twenty to twenty-four injections each). A crater was demonstrated radiographically in thirty of the forty-one patients. The previous diets of the patients were not changed and all but three of them were kept ambulatory. Thirty of the patients were relieved of their symptoms at or before the conclusion of the treatment. Fourteen of this asymptomatic group showed radiological evidence of a healed ulcer, whereas twelve showed a crater still present. Eleven showed no improvement symptomatically or radiographically. The period of observation varied from six months to one year. At the present time thirteen are still symptom-free and twenty-six have had one or more relapses. Of the twelve rendered symptom-free but in whom a crater persisted, only three have remained symptom-free, whereas of the fourteen rendered symptom-free and in whom no crater could be demonstrated after treatment, ten have remained asymptomatic. A control group of forty selected patients with acute symptoms and radiological signs of active peptic ulcer were treated with the usual ambulatory diet-alkali ulcer regimen. At the end of the first four weeks of treatment thirty-one were symptom-free. At the present time sixteen have remained symptom-free, whereas twenty-four have had one or more relapses. The clinical improvement succeeding histidine hydrochloride therapy in acute peptic ulcer appears to be symptomatic and transient. Chronicity and rhythmicity is a characteristic feature of peptic ulcer. Histidine appears to have no effect other than to alter the rhythm slightly. Histidine hydrochloride showed no constant effect on the hydrochloric acid secretion. The therapeutic indications for histidine in the treatment of peptic ulcer are necessarily limited. The extravagant claims that have been made for this substance are unwarranted.—*J. Am. M. Ass.*, 1936, **106:** 1452.

Case Reports

TWO CASES OF ENDOCARDITIS IN INFANTS

BY E. S. GIDDINGS, M.D.,

Charlottetown, P.E.I.

CASE 1

B.B., a male infant of two months, was admitted to hospital on July 19, 1935, with attacks of cyanosis and rapid, gasping respirations. The baby was the first born of twins, born on May 19, 1935, one month prematurely. Labour was normal and delivery spontaneous; presentation, vertex; position, L.O.A. The boy was of normal colour at birth, without deformity. At birth his weight was 5 lbs. 7 oz. The mother was a hard-working anæmic woman, 38 years of age, and had had no prenatal care. No venereal disease was present in the family.

At the age of two weeks the baby began having attacks of cyanosis, with rapid gasping breathing which lasted for about five minutes and then lessened. The attacks would come on about 2 to 3 times a week at first, but gradually became more frequent, until during the eighth week they occurred every two or three hours. On admission to hospital, physical examination revealed a temperature of 99° F.; pulse rate 140; respirations 40. Weight was 7 lbs. 4 oz. The colour was good at the time of admission; no icterus. The tongue was clean, throat negative, no enlargement of cervical glands.

Chest.—The respiratory movements were of abdominal type. The lungs were fairly resonant throughout; breath sounds broncho-vesicular. Coarse moist râles were heard at both apices and at left base. The heart did not appear to be enlarged. The heart sounds were clear at the apex; no murmurs. At the base the heart sounds were roughened, but no definite murmur was heard.

Abdomen.—Somewhat distended. The liver and spleen were not palpable. The genito-urinary system was apparently negative.

There were numerous petechial spots over the face, chest and limbs, and a pin-point rash over all the body. The other systems were normal. The Kahn test was negative. A blood count was not done. Urinalysis negative.

X-ray examination of the chest showed the heart to be normal in size, shape, and position. The apices of both lungs were slightly denser than the lower half. No evidence of enlargement of the thymus gland.

Attacks of cyanosis persisted and became more frequent and the baby died on July 22, 1935, at 6.10 a.m. At autopsy the lungs showed congestion and small areas of consolidation in both apices. The base of the left lung was markedly congested, its cut surface exuding blood-stained fluid. No free fluid was found in the pleural cavity. The pericardial sac contained 25 c.c. of blood-stained fluid. The heart was moderately enlarged, the myocardium a pale reddish brown, soft and spongy. No evidence of a patent foramen ovale, nor persistent truncus arteriosus. The mitral, tricuspid and pulmonary valves all appeared normal. The aortic valve had numerous vegetative growths over the valve surfaces. These were of a pinkish and yellowish colour, fairly firm though easily broken off. A culture was taken which gave a growth of *S. viridans*. Sections from the apices of the lungs showed bronchopneumonia.

Diagnosis.—Sub-acute bacterial endocarditis, with a terminal bronchopneumonia.

CASE 2

Mrs. R.P., 11-para was admitted to hospital on October 7, 1935, and delivered of twins, a boy and girl, weighing 5 lbs. 6 oz., and 6 lbs. 7 oz., respectively. Both babies were of normal colour, without deformities or blemishes.

The boy was put to the breast 12 hours after birth, being on gelatine formula, q.2.h., until this time. His weight at the end of twelve hours was 4 lbs. 15 oz. The baby appeared perfectly well until October 11th, when he refused to nurse at 6.00 a.m. His weight was still 4 lbs. 15 oz. At 10.00 a.m. he was somewhat cyanosed and the breathing harsh. Examination at this time showed nothing of note beyond cyanosis and roughening of the heart sounds. No definite murmur was noted. Temperature

was 99.8° F. at 8.00 a.m. and 99° at 12.00 noon. The baby became suddenly deeply cyanosed and died at 4.30 p.m.

The history of the parents was essentially negative. The mother had had but little prenatal care. No venereal disease was noted in the parents.

At autopsy the lungs were of a reddish colour, exuding a bloody fluid from their cut surfaces. The pleural cavity contained about 20 c.c. of fluid on each side. The pericardial sac contained about 10 c.c. of free fluid. The heart was somewhat enlarged, of a reddish-brown colour, somewhat mottled on its posterior surface. The myocardium was soft and spongy. There was no evidence of a patent foramen ovale nor of a persistent truncus arteriosus. All the valves of the heart were literally encrusted with soft friable masses of a grayish colour. The condition was such that all valves but the aortic must have been incompetent. These masses were also found along the chordæ tendineæ.

The liver and spleen were enlarged and showed venous congestion. There was no evidence of emboli. The kidneys were of a normal size and appearance.

Culture from the heart gave a pure growth of pneumococcus.

Diagnosis.—Pneumococcal endocarditis.

SUMMARY

Two cases of endocarditis in infants are reported. (1) A sub-acute bacterial endocarditis in a child of two months; onset of symptoms at the age of two weeks. (2) A pneumococcic endocarditis in a newborn infant, death occurring at the age of four days.

Both patients were males of a pair of binovular twins, a boy and girl in each pair. The other twin in each case appears perfectly healthy and is thriving.

Both of these cases were apparently of congenital origin.

SPONTANEOUS CEREBRAL HÆMORRHAGE IN A YOUNG WOMAN

BY J. H. DUNCAN,

Sault Ste. Marie

In June last a young married Canadian woman of twenty-six years was in the midst of the usual strenuous work of settling in her new little home with her husband and two healthy children. She began to have a headache, which she thought must be due to the varnish, but she worked away for nine days until its severity and vomiting forced her to give in. This pain in her head, she said, came like a wave and extended from the occiput up over the head and down her back. It made her cry out, and I found her sitting with her head supported by her hands and in great misery. Her temperature was 99° F.; her pulse rate 68; blood pressure, 115-80. She had slight albuminuria. She was admitted to the Plummer Memorial Hospital for study. Her eye grounds and cranial nerves were normal; knee jerks were equal and active; Kernig and Babinski tests positive; and it was hard for her to flex her chin on her chest. Her spinal fluid on admission was blood-tinged, as it was also on the second day, but just yellow-tinged on the fourth. She remained in the hospital for one week, always mentally alert and increasingly cheerful as she improved each day. The diagnosis on discharge was spontaneous subarachnoid hæmorrhage, and the case seemed to correspond closely to some of those described by Gordon,* of Montreal. On the fourth day after returning home she said she was fine and almost free of headache when she retired at 10 o'clock. Four hours later she suddenly passed into unconsciousness, with tonic convulsions and rigidity. Her tongue was bitten, her eyes turned to the extreme left, her pulse slow, her skin perspiring with her exertions, as she retched and was convulsed, her breathing was most stertorous. She was hurried back to the hospital where she died four and one-half hours from the onset of unconsciousness. Her spinal fluid before death was apparently 50 per cent blood.

At autopsy a large softened area was found in the right frontal lobe containing a blood clot about the size of a large egg. More clots were found in the ventricles, especially in the posterior horns and in the fourth ventricle and down the spinal cord. Besides this, blood was scattered over the base of the brain and into the cerebellum. Most of the clots were black and streaked with lighter fibrin. The large vessels at the base of the brain felt quite beaded. Specimens of brain and vessels were sent to the pathological laboratory of the Ontario Depart-

* *Canad. M. Ass. J.*, 1932, **28**: 8.

ment of Health. The microscopic examination showed a moderate degree of chronic nodular endarteritis, and the brain softening was thought to be of at least a week's duration.

This case is reported on account of the youth of the patient and because from my own observations during both her pregnancies, I know that she had a normal blood pressure. Three weeks before the headache began she had had an abscessed tooth extracted, but this probably had nothing to do with the hæmorrhage.

VENTRALLY JOINED TWINS

By C. B. CAMERON,

Petite Riviere, AND

D. A. CAMPBELL,

Bridgewater, N.S.

A young married woman, aged 18, came in at six and eight months for prenatal examination. The blood pressure and pelvic outlet were normal. One head was felt on the left side near the fundus, the other in the pelvic outlet. Diagnosis.—Twins, lying head to tail. I expected trouble, but not so much as I got (C.B.C.).

Occasional pains and leaking of water began on October 17th. On the 21st, at 8.30 p.m., labour commenced. The head, presenting right occipito-posterior, only filled the anterior part of the cervix, which was dilated to the size of a quarter. At 12 o'clock the head filled the cervix; the pains, bearing down. Morphia, gr. ¼, and atropine, gr. 1/150, were given hypodermically. At one o'clock, there was no advance. At two o'clock the pains were very severe. The morphia and atropine were repeated. The cervix was fully dilated; membranes ruptured. At three o'clock, the position was changed to right occipito-anterior. I applied forceps and delivered the head as far as the brow but could not get farther with light traction and manœuvring into the oblique diameter. Pitocin, 0.5 c.c., obstetric strength, was given. This was an error, but apparently did no harm. At four o'clock I applied full strength forceps. I was now looking for a Bandl's ring, and very, very scared. I sent for Dr. Campbell. Œdema was now noticed around the rectum. I could not get any advance.

At five o'clock I examined. I could pass my hand up posteriorly but not on the anterior side. I thought that the head of the second child was caught on the pelvis of the first. Dr. Campbell then examined. He thought that the thigh of the first child was around the head of the second. He applied traction, but could not budge things. I then went up, and got the feet of the first and delivered these easily, then the body of the first, leaving this child altogether outside the vulva. By now the upper abdomen and lower chest of each were found to be continuous and lying so as to obscure the vulva. We consulted and cut the pedicle with scissors. Dr. Campbell then went up, and got the feet of the second child, and delivered by version, then did a careful repair after delivering the placenta. Pitocin and ergot were given. The perineum was ruptured to the anus. Fld. ext. of ergot was given every four hours.

The patient had a very stormy convalescence, temperature running to 102°; pulse, 88 to 110. The vulva was œdematous. The perineal repair sloughed out on the fifth day, but the sphincter remained whole. The bladder was paralyzed, with the incontinence of retention. There was a gray slough on all the wounded parts, which lasted three days. On the eighth day the parts became clean, though an acrid discharge came on when the woman turned her face down. Her appetite remained good; the bowels moved easily with laxatives. The breasts were full and a little tender. The womb was tender until the fifth day, when tenderness disappeared. No odours were noted.

I had the bed elevated at the head, eighteen inches, gave Ergot, Ferri Quin. and Strych. Cit., gr. iii, t.i.d.; pushed fluids, and had the position changed to face downwards for fifteen minutes every four hours.

The twins were head and head, weighed five and four pounds respectively, and were joined by the lower chest and abdomen below the umbilicus, for practically the whole width of the abdomen, from the ensiform cartilage to below the umbilicus; the pedicle was about four inches long. I do not know where the cord went through. The fontanelle of the first child was pulsating when the head was delivered, but ceased before the separation.

The parents had a common ancestor in their great-grandfather. No abnormalities had occurred in either family.

The paralyzed bladder emptied nicely with pressure exerted on it as in Crédé's method, and regained its tone in a week. Three months after birth the woman has a two-inch perineal body, one deep cervical tear, no cystocele. She feels very well.

We had the bodies to keep for a while, but the parents objected and had them buried.

I know now that the proper way to have handled this case was Cæsarean section or version, but I do not think that anyone would have rejected the head in the cervix and done version.

The minister sees people at their best, the lawyer sees them at their worst, the doctor sees them just as they are. To no one more than to the physician is there offered the opportunity for service, to no one more than to him is the incentive to service more impelling. Rightly to embrace the opportunity to respond to the incentive, the physician should possess a dual personality. He should be intellectually—or, as it is oftener expressed, scientifically—minded toward the disease, but sympathetic or human toward the patient.—*The Diplomate,* May, 1936, p. 157.

A theory founded on Nature, that should bind together the scattered facts of medical knowledge, and converge into one point of view the laws of organic life, would thus on many accounts contribute to the interests of society. It would capacitate men of moderate abilities to practise the art of healing with real advantage to the public, it would enable every man of literary attainments to distinguish the genuine disciples of medicine from those of boastful effrontery or of wily address, and would teach mankind in some important situations the knowledge of themselves.—Zoonomia, Darwin.

Clinical and Laboratory Notes

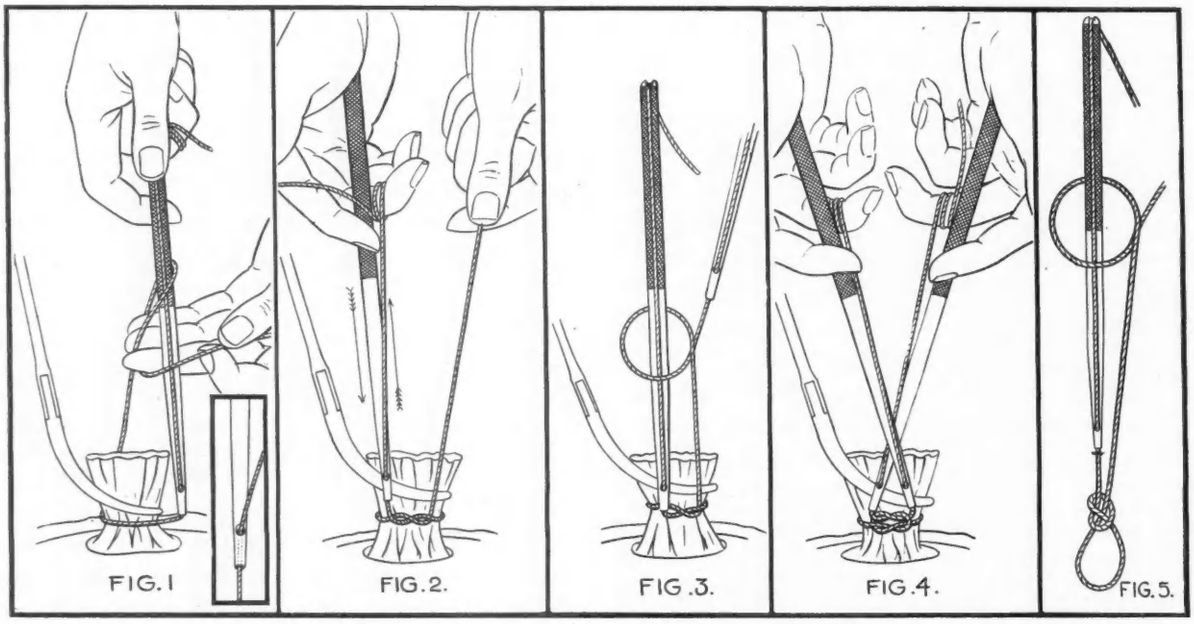
A DEEP LIGATURE TIER

By M. S. Hollenberg, M.A., B.Sc., M.D.,

Winnipeg

The tying of a surgical knot in a deep cavity has always been a source of difficulty. The knot tier to be described consists of a metal rod tapered to a blunt point with the eye in the point emerging on the side of the taper. This tier enables one to tighten the knot on its own plane.

The ligature is threaded through the tier as illustrated in the inset (Fig. 1). Figs. 1 to 4 demonstrate the method of tying a square knot. The first tie is made by crossing the hands carrying the ligature around once and leading the tip of the tier downward while tightening the threaded end of the ligature (Figs. 1 and 2). The tip of the ligature carrier holds the first tie so that it cannot slip. By fixing the threaded end of the ligature in the V placed on the distal end of the tier the first tie is fixed. Fig. 3 shows the second tie being made around the instrument and being brought down with a second knot tier (Fig. 4). As the knot is tightened the ends of the tiers are crossed. As many ties as desired may be added in a like manner. Fig. 5 shows a simple and rapidly executed knot. The fixed end of a slip-knot is threaded through the tier, and when the noose has been guided to the point of ligation the free end is pulled taut. A "half-hitch" is now placed around the tier, led down, and tightened above the slip-knot. Many of my friends and myself have been using this tier with success for the past six months.



Regarding this subject of Rest in its highest, closest, and best relation to mankind, and looking at it by the aid of my feeble penetration, I would, in all humility, remind you that when God ordained that man should live by "the sweat of his face," as a punishment for his disobedience, it pleased Him, in the plenitude of His unspeakable benevolence, to permit man's fatigue and temporary exhaustion to be followed by his greatest earthly solace, the blessing of rest and repose, by calm and peaceful sleep; a blessing which should be the immediate reward of his labour. Nature devoting her best efforts, during this period of rest and sleep, to repair those powers which may have suffered exhaustion, to renovate the bodily strength, and to restore the mental vigour, mitigates man's punishment by a source of real and refreshing enjoyment, enabling him to resume his labour in all the delightful vigour of a renewed existence. —On Rest and Pain, Hilton, p. 2.

Editorial

THE NERVOUS AND MENTAL SEQUELÆ OF CARBON MONOXIDE POISONING

CARBON monoxide poisoning occurs with sufficient frequency, especially during the winter months, to rivet everyone's attention upon it. Naturally, the cases which end fatally are the most dramatic, and the immediate and fulminating effects of the gas may be said to be well understood, but it is not so widely known that, short of a speedy lethal result, striking sequelæ are occasionally met with, sometimes a little delayed, among which certain related to the central nervous system are of special importance. In this connection we would cite a valuable paper by Drs. T. E. Dancey and G. E. Reed, of the Protestant Hospital, Verdun, Que., which is worthy of attention (see this issue, page 47). These authors report two cases in which striking mental symptoms followed exposure to carbon monoxide, in the first from the exhaust of a motor car, and in the second from coal gas. The first patient, a girl of nineteen, one week after exposure, became violent, began to talk peculiarly, and developed delusions and hallucinations. There was rigidity of the muscles on movement, and the tendon reflexes were exaggerated. During the fifth month of her illness atrophy of the retina about the optic discs was discovered. At the time of reporting the patient was greatly improved. The second patient was a man of sixty-six. He was seen four weeks after exposure, and then was confused, quarrelsome, and resisted dressing, feeding, examination, and care of his person. On physical examination there was marked pallor and œdema of the lungs. There was cog-wheel rigidity of the elbows and knees on flexion. The tendon reflexes were much increased. There was definite intension tremor after one month. Two months later the man had practically recovered. The authors remark that their first case on casual examination resembled hebephrenic schizophrenia in a juvenile type; the second resembled general paresis. They note, also, as have others, that the manifestations of carbon monoxide poisoning are multiple and divers, and state that symptoms have been described as initiating such conditions as hemiplegia, amnesia, localized paralyses, paralysis agitans, disseminated sclerosis, complete dementia and multiple neuritis.

So far as can be gathered from the published reports the percentage of cases of nervous and mental sequelæ is not high, although in the aggregate their number must be considerable. Petersen[1], of Copenhagen, states that in his hospital, from 1900 to date, about 1,400 cases of carbon monoxide poisoning have been treated, and in only five or six of them were serious late sequelæ observed. One of the patients concerned developed mental symptoms which promised to be permanent; another, one year after being poisoned by carbon monoxide in a garage, was still tired, drowsy, and apathetic, hardly able to do a day's work, and very forgetful, though previously he had been energetic and enterprising.

More extensive observations have been made by Shillito, Drinker, and Shaughnessy[2]. Their information was obtained from a study of the records of seven state mental hospitals serving the metropolitan area of New York City covering a period of ten years. During that time there were at least 21,143 admissions for acute carbon monoxide poisoning. Among these were 39 certain cases of nervous and mental sequelæ. Such patients formed 0.05 per cent of the total admissions. Thus it appears that serious mental and nervous symptoms are infrequent in relation either to other nervous and mental diseases or to the total number of acute exposures. These observers found that when nervous

1. PETERSEN, S.: Carbon monoxide poisoning and its sequels, *Nord. Med. Tidsskrift*, 1935, **10:** 1507.
2. SHILLITO, F. H., DRINKER, C. K. AND SHAUGHNESSY, T. J.: The problem of nervous and mental sequels in carbon monoxide poisoning, *J. Am. M. Ass.*, 1936, **106:** 669.

or mental damage occurred they did so in cases where the poisoning was extreme. Complete unconsciousness was the rule and the most active resuscitative measures had to be instituted. It is worthy of note that none of the cases followed exposure to small doses of carbon monoxide extended over a long period of time. In one-third of the cases a clear interval of from seven to twenty days preceded the onset of the special symptoms; in two-thirds the symptoms set in immediately after the exposure. The mental symptoms consisted of confusion psychosis, disorientation, loss of judgment and amnesia. Motor excitability and asphasia were much less frequent. Hallucinations, delusions, or convulsions were not conspicuous. The nervous sequelæ consisted of signs varying in intensity, from slightly increased deep reflexes to well advanced Parkinsonism. Sensory disturbances, such as superficial anæsthesia and peripheral motor neuritides, were also met with. The cases all showed improvement, but the final result depended, naturally, on the amount of the initial damage. Of the total groups of 43 patients 23 recovered completely, 9 suffered permanent nervous or mental damage, and 11 died.

Paul Michael[3] comments on the widespread dissemination of the pathological lesions. No tissue or organ is exempted, hence the bizarre picture we sometimes see. He refers to the fact that all changes are based on vascular degeneration due to anoxæmia, which results in parenchymatous degeneration. In experimental animals bilateral softening of the basal ganglia, degeneration of the ectomesodermic barrier, œdema of the brain, and degeneration of the peripheral nerves are found. Other lesions are myocardial degeneration, pulmonary congestion, with localized hæmorrhages, parenchymatous degeneration of the liver, and degeneration of the skeletal muscles.

Dancey and Reed agree that an endless variety of nervous and mental symptoms may follow carbon monoxide poisoning. There are no lesions in the brain which are typical and invariable, which is probably the reason for this. The amount of damage seems to depend on the age of the patient, the quality of the gas inhaled, the duration of life after the exposure, the amount of degeneration of the vascular system, and whether or not there was pre-existing disease of the brain. The following lesions have been described—degeneration of the ependymal cells of the choroid plexus; swelling of some cells and shrinkage of others; minute hæmorrhages throughout the brain; demyelinization of the white matter; softening of the cortex, especially in the infra-granular layer. The olfactory apparatus may be involved, and even the cells of the anterior horns of the spinal cord. The theory of vascular degeneration from anoxæmia, with the resultant hæmorrhage and parenchymatous damage, would seem to explain satisfactorily the variability of the lesions. A.G.N.

3. MICHAEL, P.: Chronic carbon monoxide poisoning—a present day hazard, *Calif. & West. Medicine*, 1934, **40**: 19.

SHOULD AN INTERNSHIP BE REQUIRED TO OBTAIN A LICENCE TO PRACTISE?

A VERY few recent graduates in medicine nowadays go directly into practice without having had the benefit of a hospital internship. Whereas in a former generation only the fortunate few had this privilege, the student of today considers an internship to be an essential part of his training, and an ever-increasing number are taking not merely one year but two, three, or even more years of hospital training. If one includes those few who are definitely planning for laboratory or research work and go at once into the laboratory departments, it frequently happens that 100 per cent of the graduating class proceed to take an internship. So important is this aspect of clinical training that the internship year must be completed before graduation at Manitoba, Dalhousie, and Montreal, is required to a certain extent at Queen's, and has recently been introduced at McGill.

If the value of the internship year is thus so widely appreciated, and if the vast majority of our recent graduates voluntarily elect to take this training, has the time come when the licensing bodies in Canada should

take under serious consideration the advisability of making the internship year obligatory for licensure? It is not suggested that the internship year should be of necessity part of the medical course—that is a matter for decision by the medical schools—but, should a minimum of internship experience, either before or after graduation, be required by our licensing bodies before certifying applicants as prepared to enter practice?

Although certain of the medical schools do have an internship year, internship experience is not now obligatory for licensure in any province. From replies received it would appear that the examining bodies are not concerned with the internship training of the applicant as such, but it is insisted that any undergraduate internship should not have interfered with the academic training. The Medical Council of Canada is governed to a considerable extent by the standards maintained by the provincial licensing bodies, for "the standard of examination shall not, at any time, be lower than the highest standard for the like purpose then established for ascertaining the qualification for registration in any province". Hence there is no internship requirement.

Our standards of qualification have been based primarily upon the British tradition, which has emphasized the academic rather than the practical aspect of preparation. Across the border the desirability of a practical training, as acquired in an internship, is being expressed in their requirements for licensure. The National Board of Medical Examiners, whose certificate is now recognized by forty-three states, requires "completion of a contract of at least one year in an acceptable hospital as an intern, or a year in an acceptable laboratory". For individual state qualification it is required in nineteen states, including Illinois, Iowa, Michigan, New Jersey, Pennsylvania, Washington, Wisconsin and the District of Columbia. Fifteen medical schools require completion of an internship before a diploma is awarded; this, however, is but indirectly related to the question under discussion.

In several provinces the Registrars have reported that the desirability of requiring either an undergraduate or a graduate internship has been discussed, but no immediate action is being contemplated. Obviously no person desires to add to the difficulties of the young graduate; to many students financing their courses on a "shoestring" the non-remunerative years are long enough, and an internship, even with maintenance and honorarium as paid in many hospitals, would be a real effort. However, such men going directly into practice are greatly handicapped in treating their patients, and, with practically all of their contemporaries favoured by internship training, are going to find it increasingly difficult to hold their own in an ever-tightening competitive field. Only a small number would be affected anyway. Several Registrars reported that for some years practically no men had been registered who had not had internships, and the step from a voluntary arrangement, followed almost by 100 per cent, to an obligatory one would be much less of a possible handicap than was that of adding a year to the course some years ago.

Of course it would be necessary to ensure that the internship be taken in a hospital acceptable to the licensing bodies. Fortunately we now have in Canada a large number of hospitals approved for intern training by the Canadian Medical Association and these hospitals can absorb for training more than the annual output of the medical schools. A number of graduates take internship in the United States, but the American Medical Association list of hospitals approved for internship permits ready check of that training. Some arrangement would be necessary, also, to place proper evaluation upon the work of those who go to Great Britain for post-graduate study. The comment of one Registrar may not be without significance: "Personally I am very much in favour of the proposal of requiring internship, and I believe that the day is not far distant when it will be impossible for a man to register in any province in the Dominion without having gone through at least one year of internship."

G. H. AGNEW.

Editorial Comments

The Problem of Sciatica

We would draw attention to the paper by Dr. J. A. Nutter in this issue on the subject of sciatica. As Dr. Nutter clearly points out the etiology of chronic sciatica is obscure and its treatment therefore correspondingly limited. It is shown that several theories of its causation have been brought forward, but none have received lasting support. At the moment the tendency amongst some orthopædic surgeons is to attribute the pain of sciatica in some cases to undue muscular and fascial tension in the region of the hip. Just how this serves to produce the symptoms has not been explained very clearly, but acting on this assumption a good deal of surgical work has been directed towards partial stripping of the gluteal muscles from the ilium, or cutting of the ilio-tibial band; and with good results.

Dr. Nutter has followed up this line of attack but his observations lead him to think that the effects of this operative work take place mainly in one region, namely, where the sciatic nerve passes out from under cover of the overlying piriformis muscle.

From the anatomical point of view this is a site at which the nerve is quite liable to pressure, and if, as Dr. Nutter found in some instances, there should be at this point a thickening of the fascial covering of the muscle in the form of a fibrositis, then the tendency to compression of the nerve is that much increased. In some of the cases reported, all that was necessary was to incise or tear through such fibrous thickenings, which were occasionally definitely band-like in nature; in others it was felt that the ilio-tibial band also required incision; at any rate the relief of tension was obtained.

The results of this work have been encouraging and we look for further observations on the subject. One point of note is that the operation for incising such fibrous bands is not at all difficult. In intractable cases of sciatica it deserves careful consideration. H.E.M.

Dr. Campbell Palmer Howard

It is an all too common and sad experience that the very characteristics and qualities which render men great often contribute to their untimely end. So it was with many heroes in the War; soldiers, whose gallantry, courage and devotion to a cause led them to be exposed to dangers and often to death. So it was with Campbell Howard, who never flinched when duty called, whose loyalty and conscientiousness impelled him so recently at great personal risk to carry out his obligations to the medical profession of California, where he had undertaken to give a series of lectures and clinics. Having accepted this invitation his sense of duty impelled him to take the risk involved in an apparently subsiding attack of deep phlebitis. It was still simmering when he entrained for the Coast. His clinics were enthusiastically received. The impression he made in the West was profound, as has been fully expressed in a number of letters received from colleagues who heard him. He had done his duty—an arduous one in the circumstances—but the task was too much. Exacerbation of the phlebitis occurred and a clot broke away causing pulmonary embolism and death. He died as he had lived, placing his duty before personal consideration and doing his task well.

Few men have embarked on a career in medicine with a better background than did Campbell Howard. Himself the son of a distinguished father, one of the outstanding men of his time in this country, he enjoyed a notable and successful career. Early after graduation at McGill he became an assistant of Osler's at Johns Hopkins Hospital and absorbed the inspiration which came naturally to all those who enjoyed that privilege. To the qualities of an excellent clinician and teacher was added an investigative mind. He knew the value and the importance of modern technical methods of the laboratory, and incorporated them in his clinical work. He was never carried away, however, by the ultra-scientific attitude which disregarded the patient in favour of a syndicate of laboratory reports. While fully appreciating the value of investigative laboratory methods, he always kept his feet on the ground and was a sound clinician. His very natural human instinct, too, contributed in no small measure to his success as a practitioner. The example of Osler was ever before him, and the advantages of that intimacy were evident in his every day intercourse both socially and professionally. His success as a Professor of Medicine in Iowa University had not only ranked him high on the continent, but attracted to the clinics doctors and patients from afar. Howard was the leading consultant in the State of Iowa. When, after some years, he was invited to occupy the Chair of Medicine at McGill and assume the Directorship of the Department of Medicine in the Montreal General Hospital, his acceptance remained for some time a matter of doubt, and the decision was difficult, for other leading medical schools of the United States were approaching him. Ultimately, however, his devotion to British institutions determined his choice of McGill.

His investigations on cirrhosis of the liver and the relation of liver treatment to cases of anæmia were coincident with similar important work being carried on elsewhere. Had it not been that other investigators ante-dated his results by a very short period, his name would have gone down to history as one of the great discoverers in his profession.

His work at McGill University and at the General Hospital was characterized by thoroughness and conscientiousness that added not a little to his diagnostic acumen. His interest in the literature was wide, and his understanding of the human side of practical medicine made him beloved by all his patients. His many contributions to the literature were characterized by sound commonsense, and were based on a wide experience that rendered his monographs conspicuously valuable to practitioners and teachers. He spoke and wrote with an authority that was founded on experience.

Dr. Howard will long be remembered by his colleagues and students, not alone because of his teaching ability and his skill in practice but as the very highest type of professional gentleman. C.F.M.

Manitoba and the Chiropractors

During the recent session of the Legislature of Manitoba a bill was introduced providing that chiropractors be licensed, that they be empowered to determine the qualifications required for the profession, and that they be entitled to carry the title of Doctor of Chiropractic. The debate on the second reading of this bill was the best heard in the legislature for many years, and cut right across party lines. The bill was introduced by a government supporter and supported by Government, Labour and Conservative members, but it was opposed by other government supporters, by the Minister of Health and Public Welfare, the Conservative leader, and by a Labour member. All the ministers present voted against it, and it was stated that the premier, who was absent, also opposed the bill. The debate was fought out on the question of whether the interest of the public of the province would be furthered by the passage of the bill and it was conducted on a high plane throughout. The bill was thrown out by a vote of thirty to thirteen.

In his column, "Under the Dome" the *Winnipeg Free Press* political writer made the following comment: "If the Legislature had passed this bill Manitoba would now be committed to the proposition that scarlet fever is not infectious and can be cured by thumping the patient's vertebræ. It would now be the law of the land that appendicitis or diphtheria disappears if a chiropractor touches the right spot, from which it follows (if there is any logic in public affairs) that there would be no sense in any further effort in these parts to train surgeons or prevent epidemics. The Legislature by the satisfactory margin of 30 to 13 rejected this disastrous nonsense".

Though the majority against the bill was decisive, there was no assurance when it was first introduced that it would be defeated. The United Farmers of Manitoba and the United Farm Women of Manitoba had passed resolutions in favour of the chiropractors, and these bodies carry much weight with rural legislators who form a majority of the House. A prominent member of the Legislature is himself a chiropractor, and members were flooded with literature supporting the claims of the chiropractors. Credit for overcoming this adverse situation must be given chiefly to the Legislative Committee of Twelve, a group of doctors representing equally the College of Physicians and Surgeons, the Manitoba Medical Association, the Winnipeg Medical Society, and the Faculty of Medicine of the University of Manitoba. The chairman of the committee was Dr. Gordon S. Fahrni. Members of the committee twice interviewed the Premier, the Minister of Health and the Minister of Education. The Board of Governors of the University protested strongly against the passage of the bill, which would give government sanction to men with little preliminary education and scant training even in their own field. The Manitoba Hospital Association also entered a protest against an act which would leave hospitals wide open to infection. The Committee of Twelve strove constantly to keep before the legislators the fact that the issue was not a struggle between the doctors and chiropractors, between those that enjoyed a monopoly of the healing art, and those that wished to break through this monopoly, but that it was the duty and responsibility of the legislators to decide how the bill would affect the health and welfare of people of the province as a whole. By their long-continued efforts the Committee of Twelve succeeded where success had seemed hardly possible, and in so doing deserve the gratitude of the people of Manitoba.

ROSS MITCHELL

Special Article

THE LESSONS OF RACIAL HISTORY*

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There are many phases of racial history, and the study of any of them will throw new light upon the nature of human events and of human conditions. On the present occasion however we shall only be able to touch upon one such phase, and perhaps learn a lesson or two from it.

All students of anthropology, all small children, and most simple-minded people are able to recognize that there are differences and distinctions between human races. There is however a section of what is called the "intelligentsia" which insists that no such differences exist, and that even to make any distinctions between black and white races, for example, is not only the sign of a prejudiced mind but highly unscientific. The story was once current of an Irish priest who went to Rome and succeeded in convincing the Pope that black was white—at least the Pope was unable to refute his argument. But even the mental agility of the Irish mind could scarcely succeed in convincing most people that all races are alike, and that no distinctions are to be made between them.

Not only are the distinctions between the four or five great racial divisions of humanity both clearly evident and of obvious significance but such distinctions are just as readily made, and no less significant, between the major subdivisions of these primary races. We need not here go into an analysis of these racial distinctions, but let us first note some of their effects upon racial history.

The whole of civilized history for the last twenty-five hundred years may be seen to have been fundamentally a struggle between the Asiatic and the European races. In rare instances the European races have been the aggressors. But much more often it has been the Asiatic races that have tried to over-run Europe and to make Europe Asiatic. Alexander's conquest of Persia and India was essentially a counter-offensive to remove the Persian menace to the Grecian world. Likewise, the Roman penetration into Asia was essentially protective in its nature. And the Byzantine Empire, which it has been the fashion to consider effete and degenerate, was almost constantly at war with the Saracens and Turks for eight hundred years, throughout which period it succeeded in protecting Europe from Asiatic invasion on its own frontiers. Then there was the attempt of the Huns under Attila in the 5th Century, which was stopped at Chalons by the Visigoths, that of the Saracens and Moors in the 8th Century, which was stopped at Tours by the Franks under Charles Martel, and that of the Mongols under Badur, the son of Otocai and grandson of Ghengis Khan, in the 13th Century, which was stopped at Liegnitz by the Teutonic and Polish knights under Prince Henry of Liegnitz. In all of these instances the penetration of these Asiatic aggressors had been far into western Europe, and if these attempts had not been defeated Europe would have become Asiatic, and Aryan and Christian civilization would have perished then and there. And we should not be here today. Thus we may see that the major issue in European history has been a racial issue.

These long past events might not seem of immediate interest or importance to many of us, but they teach us that every few generations Asiatic races may be likely to renew this attempt. And unless the pacifists can work a profound change in the nature of all of the Asiatic races, such attempts may be expected to recur in the future. It would look as though the next assault of Asiatic races upon Aryan civilization would most likely come from across the Pacific, and it is not at all difficult to foresee and visualize the possibility that the White Race in the Western Hemisphere will one day need to fight with all its resources for the survival of its civilization, and of itself. It is quite evident that it is only by the anticipation of this eventuality and by a thorough and concerted preparation for it that the White Race of the Western Hemisphere can hope to resist it successfully when it arrives. Indeed it is only by a thorough and concerted preparation, a thorough state of defence, a sufficient man power, and a united front of the white races, that such an eventual assault might possibly be forestalled and discouraged.

But let us go back and examine the racial conditions and racial history which have a fundamental bearing upon this future problem. It is generally agreed among anthropologists that the white races of European origin are made up of three different primary racial elements that have been indigenous to Europe from the earliest times. These three indigenous racial elements are known as the Nordic, the Alpine, and the Mediterranean. Very briefly described, the Nordic is long-headed, tall, and

* An Address given before the Rotary Club at Brantford, February 18, 1936.

blond; the Alpine is round-headed, stocky, and mildly brunette; and the Mediterranean is long-headed, of slighter and shorter stature, and dark brunette. From prehistoric times the Nordics inhabited Northern Europe; the Alpines, Central Europe; and the Mediterraneans, Southern Europe. And while there have been innumerable mixtures and blendings between them, these three racial elements are each still predominant in these respective areas of Europe.

These three European racial elements not only differ in their physical qualities but in their mental make-up. Consequently they tend to hold somewhat different attitudes, to react differently to the various problems of life, and to develop their own distinctive racial cultures. These respective cultures, however, are reconcilable and supplementary to one another rather than antagonistic, and hence these racial elements find that they can live together in accord and with mutual advantage.

The term "Aryan" has been used as a convenient expression to include these three racial elements and all mixtures between them, from which few of this composite race are entirely free. This term however is a linguistic rather than a racial term, but it serves to embrace all of these racial elements, inasmuch as it applies to the group of kindred Aryan languages which practically all of them speak.

Those who have their own reasons, however, for deprecating and opposing any recognition of racial distinctions passionately assert that such distinctions are made not on any factual or anthropological grounds but solely from the invidious sense of race-consciousness and from racial antagonism and prejudice. These opponents are particularly incensed and ironical over the term "Aryan", which goes to indicate the cultural unity of, as well as a certain mixture between these three white racial elements, and they are fond of hurling the jibe that those who call themselves Aryans do not themselves know what they are. Sir Arthur Keith has given a trenchant answer to this by saying that Aryans may not always know just what they are, but they well know what they are not. "They are not Chinamen, they are not Arabs, they are not Jews, they are not Negroes, and they are not American Indians."

But these opponents are no less incensed over the term "Nordic", and they try hard to create a popular prejudice against it by distorting its true application, knowing that the popular knowledge of racial distinctions and terminology is by no means general nor accurate. Thus the attempt is made to create the impression that the term "Nordic" is essentially a perquisite of the Germanic racial group which it applies to itself for its own aggrandizement, and that any others that may wish to identify themselves as of Nordic stock only do so out of their desire for self-approval and self-admiration. But only to show the important, and, we may judiciously say, vital, part that the Nordics have played in racial history, let us get at a few facts that go to distinguish the Nordic racial stock. The Nordic race of Europe is the one human race that has developed pure blondness in its biological evolution, namely, the all but complete absence of pigmentation of skin, of hair, and of eye colour. All other racial elements of the human species have a distinctly greater pigmentation. The original proto-Nordic racial stock, from which all Nordic stocks derive their major inheritance, was unquestionably of such an unpigmented character. It would be strange indeed, and contrary to genetical observation and experience, if this racial stock did not possess and transmit other racial qualities and traits that were equally identified with it and equally characteristic.

The earliest known traces of the Nordic racial stock in Europe are from Nordic remains in Scandinavia that can be definitely dated at about 12,000 B.C. By about 2000 or 1500 B.C. Nordic tribal groups had penetrated to different parts of Europe, and in many instances displaced, or dominated and mixed with a precedent population of Mediterranean racial stock. Also at about that period one Nordic stock penetrated to and conquered India, and established the system of caste, in order to prevent their mixture with the indigenous Negroid stock, an effort which, we should note, failed in its purpose. And today none of those, even of the highest caste in India, are free of such mixture. But more and more evidence has accumulated from archæological discoveries as to the extent of early Nordic penetration into other parts of Asia.

At this period, of about 2000 or 1500 B.C. a large part of present-day Germany, the North Sea littoral, the whole of France and of Northern Italy, and a part of Spain were occupied by a number of kindred Nordic tribes, known as Gauls or Kelts, which names, along with that of "Gael", all spring from the same word-root. Thus there are the many references in Roman literature to Gauls, to cis-Alpine Gauls, and to the Kelt-Iberians in Spain. It is of some interest in this connection that the Greek legend of Elysium is traceable to a tradition of early Greek adventurers, who described a country in Spain called *Elision*, inhabited by a Keltic race, so gentle and so kindly that life among them was one of unruffled happiness and contentment. Today there is a Galicia in Spain and a Galicia in Austria. There was an ancient Galacia in Asia Minor, and there is still a Galilee in Syria and a Galata quarter in Constantinople.

The Kelts were one of the earliest Nordic stocks that developed a racial urge for expan-

sion and conquest, hence their wide distribution over Europe at this early period. But there were other Nordic racial stocks that manifested this racial tendency not so long afterward. Among these were the Achæans and the Ionians who descended from the Dacian plains in Central Europe between 1500 and 1200 B.C., conquered Greece and its Mediterranean population of Pelasgians and founded the Greek civilization, and the Latins and Oscans who came from the same region somewhat later conquered the Mediterranean population of Italy, and founded the Roman civilization.

Still another Nordic stock, of whose early movements our knowledge is more meagre, manifested this tendency, possibly at a still earlier period. This was another kindred group of tribes, generically known as the Scythians, of whose settlements there is archæological evidence as far west as southern Germany, who spread over southern Russia and south-eastern Europe in prehistoric times, who were well identified with the ancient history of that region, and after whom an area to the north and east of the Black Sea was called "Scythia" by the Greeks.

We further know from historical records that Sargon, king of Assyria, conquered Palestine about 700 B.C., and in order to secure his frontiers expelled the Semitic population of northern Palestine and colonized it with Scythians and Gauls, who had been his soldiers. There are also references to Sargon using Goths as soldiers, so it might appear that he was partial to Nordics as fighters. There is not only a Scythopolis in Galilee, itself a Nordic name, but many other place-names in it are linguistically Aryan or Nordic. This and much other historical data leave practically no doubt that the population of Galilee, among which Christ was reared, and from which his early followers came, was essentially Nordic. There is an age-long and widespread tradition among the population of southern Russia, which is very largely of Scythian descent, that Mary, the mother of Christ, was a Scythian. It might also be remarked in this connection that the tradition that Christ had fair hair and blue eyes dates from the Second Century, when there were people alive who would have known those who had seen Christ.

And it might well be regarded as a fact of the deepest racial significance that the whole Aryan population of Europe was promptly converted to the teachings of Christ because these teachings were congenial to its racial spiritual nature, whereas, despite the assiduous and continuous efforts for 1900 years to convert other races to these teachings, these efforts may be said to have all but failed. Hence Christianity is essentially an Aryan religion. Thus we may get a fairly adequate picture of the Nordic influence upon our civilization in its prehistoric beginnings and in its early formative period.

But let us return to the early Nordic developments in western Europe, where we left the Kelts and probably some Scythians in possession about 1500 B.C. Their occupation of the territory into which they had spread was not to be undisputed. Between 1500 and 1000 B.C. another Nordic group, the Germanic racial stock, now felt the impetus for expansion and conquest. This Germanic stock, of which there were many tribial divisions, soon occupied most of what is now Germany and Austria, and the North Sea littoral, displacing the Kelts and Scythians who were there before them. In such tribal or racial conflicts it would seem to be an invariable rule that the vanquished group only has the alternative of complete submission or migration in order to escape extermination. For example, the American Indians were being gradually exterminated as long as they offered resistance to the whites. It was only their eventual submission that prevented their total extinction.

It might be inferred that the Scythians resisted until they were practically exterminated in western Europe, or, possibly, there may have been migrations to the eastward of which we have no trace as yet. There are however still some remainders of this pre-Germanic Keltic stock in the highlands of Germany and Austria. While we know nothing of the ins and outs of this early Kelto-Germanic conflict, the migration of that period of the Kelts who occupied the North Sea littoral to Great Britain and Ireland might seem to have been the result of the pressure upon them by this Germanic assault. But for this migration, however, the subsequent racial composition in Great Britain, and particularly in Ireland, would have been very different.

Until well into the Christian era there were many similar struggles between Nordic groups in Europe for domination and territorial expansion. Perhaps the most notable of these, particularly in its final effects upon our civilization, was the invasion and conquest of England by the Germanic tribes of the Angles, Jutes, and Saxons, some of whom had previously displaced the Kelts on the North Sea littoral who had migrated to England, and whom they now followed. Then, there were the epic exploits of such intrepid and restless Nordic adventurers as the Goths, the Vandals, and the Franks, who imposed their domination upon almost the whole of western Europe and determined its ultimate political divisions. Among the great number of these incursions, the Vandals, for example, penetrated through Spain to the coast of Africa, and ruled a large extent of it for a hundred years. They paused on their way long enough to give their name to Andalusia, or Vandalusia. Later on the Crusades offered all these restless Nordics an outlet for

their spirit of adventure and conquest, and at the same time enlisted them in the same cause, rather than against each other.

Toward the end of these marchings and counter-marchings of Nordics over Europe, the Nordic spirit of racial adventure and expansion began to fructify in the Scandinavian Nordics, and manifested itself in their expeditions to Iceland, to Greenland, and to America, and in the numerous descents upon Europe of the Danes and the Norsemen, in which among other things the Normans conquered Sicily, where they established a kingdom which endured for a hundred years, and England, where their tenure was to be permanent.

Thus the outstanding events of European history have been the constant efforts and struggles on the part of Nordic racial elements to conquer and dominate every region and country of Europe, sometimes to conquer and dominate other racial elements, and sometimes one another. Indeed, this history might be regarded as essentially an epic of Nordic enterprise and adventure, often of bold aggression, but at other times of heroic resistance and defence. Some no doubt may be impatient with this recital of racial facts, and hasten to denounce it as an egregious Nordic boast. But in this instance it has a much more serious purpose, namely, as to the important lessons that may be derived from such racial facts.

Quite as distinctive a racial trait of all these Nordic stocks as their blondness was their irrepressible enterprise and their great bravery and prowess in warfare. Racial history is full of examples in which racial groups, not only by their prowess but by their employment of improved and more effective modes of warfare, have increased their racial dominance, and have enhanced their power and their prospects of survival. It was the Nordics, for example, who introduced the horse and the war chariot into ancient warfare. The horse apparently was unknown to other races before they came into contact with the Nordics. In Egypt, for example, the horse was unknown until the invasion of the Hyksos about 1500 B.C., and Thotmes IV, of the 18th Dynasty, was the first Pharaoh known to use horses and a war chariot. The centaur myth is a Pelasgian myth, obviously originating in the first glimpses that these early inhabitants of Greece had of their mounted Achæan foes, by whom they were soon to be conquered.

Throughout the Dark and the Middle Ages, and up to the invention of gunpowder and firearms, by far the most formidable of all warriors were the mounted Nordic knights, whose code of honour was no less thoroughgoing than their bravery. These knights were always of the upper, or ruling, class, and the ruling classes in all countries of Europe in those days, as well as since, were Nordic. Christian burials of these times invariably show these knights to have had long Nordic skulls, and most of the priests who were buried within the chancel had round Alpine skulls. Thus racial history abundantly proves that Nordic stocks have always excelled other races in their fighting qualities and ability, and that the position that Nordic stocks have occupied among other races is largely due to this congenital racial quality.

These different Nordic strains, notably represented at present by the Keltic, the Anglo-Saxon, the Germanic, and the Scandinavian racial stocks, can be seen each to possess particular racial qualities in which they excel, and they likewise have their particular racial weaknesses which they would do well squarely to recognize and take into full account. It is these racial qualities that are peculiar to each of these Nordic groups which serve clearly to identify them and to distinguish them from one another. But they likewise have many racial qualities in common which not only distinguish them collectively as members of the great Nordic stock, but which furnish an ample basis for their mutual understanding and cooperation. First and foremost they are all inspired to strive for freedom, a freedom which they are not over-disposed to abuse, and which they are generously inclined to concede to others. Likewise they are all indomitable races; none of them know what complete submission to domination is and they will resist it as long as they survive. The Nordic Kelts of Ireland, for example, no doubt largely derive their distinctive racial character from their mixture with the antecedent racial stock which they conquered and dominated. But they fully share with other Nordic races the quality of being indomitable. Six hundred years of being ruled by another Nordic racial stock did not suffice to gain their submission. The lesson to be learned from this and from other such events in racial history is that one Nordic stock cannot hope to subdue another. It might exterminate it but it could not subdue it. It is this indomitable racial spirit that has made the Nordic stocks the great fighting stocks of Europe and likewise the dominant governing element in every country of Europe. But of much greater import is the fact that it is this indomitable racial quality of the Nordic races which has saved Europe and Western Civilization from becoming Asiatic. It was Nordics in the first instance who created the Greek and Roman civilization upon which Christian civilization is founded. It was Nordic Visigoths who saved this Christian civilization from the Huns at Chalons. It was Nordic Franks who again saved it from the Saracens at Tours. It was the Nordic knights who saved it from the Mongols at Liegnitz. And it was the Nordic Varangians and armies of other Nordics by which the Byzantine Empire protected

Europe for 800 years. The obvious lesson of all this that in such a future crisis the result may conceivably be otherwise for Christian civilization if there are not sufficient Nordics left to defend it when such a crisis shall arise.

For this reason alone, European nations need above all things to avoid internecine wars, which have been responsible for the great diminution of the Nordic element in them. For example, de la Pouge, the great French anthropologist, estimates that France was 75 or 80 per cent Nordic in the time of Louis XIV, and that it is now less than one-third of that. There is strong ground for the thesis that no European war of modern times has resulted in other than ultimate loss and injury, even to the victors, a thesis which Nordics would do well to ponder. And as for Nordic races fighting one another they need well to learn the lesson that any lasting domination of one another is impossible. They need also to learn that their common racial qualities are a sufficient basis for mutual understanding, and that the confidence in one another's honour and fair-dealing will seldom be betrayed. And they need above all things to beware of the subtle efforts that are constantly made to bring them into conflict with one another. But the way to invite, rather than to avoid, war is by the pacifist formula of refusing to bear arms and abandoning all defensive measures, and then freely indulging in uninformed and gratuitous criticism, and impugning the honour and good faith of other national groups. In personal contacts such behaviour usually results in a black eye, if not in more serious consequences.

But the phase of this racial history which most nearly concerns us, and from which we might learn the most important lessons, is that which pertains to the Anglo-Saxon and Norman Nordic stocks. It was their dauntless Nordic spirit of adventure and expansion which prompted the Angles, the Jutes, and the Saxons, and later the Normans, to cross the North Sea and seek a new habitat for themselves in England. And it was this same intrepid Nordic spirit which again prompted these Nordic stocks a few centuries later to expand and spread from this insular habitat, and to subdue and occupy an entire continent in the Western Hemisphere, and yet another in the Antipodes. It was such manifestations of the Nordic spirit which served to place the Anglo-Saxon branch of the great Nordic race in a commanding position in the human world, and in the evolutional progress of the human race.

This is our heritage from these Nordic forebears. Do we desire to retain it, and do we desire to perpetuate the racial qualities and traits which they bequeathed to us? Do we desire to preserve the free institutions and the moral standards which can only arise from such racial qualities, and which cannot survive in their absence? Are we ready to apply our full effort and to employ every means of safeguarding this heritage? Are we ready to carry on the great racial enterprise which our forebears began, to the fulfilment of the great racial destiny which their courageous action made possible to us? Or do we wish to resign this task? We can only give one answer, unless we ourselves have lost the racial qualities of the Nordic stock from which we sprang.

THE MANTOUX TEST IN CHILDREN.—G. G. Kayne discusses the Mantoux test in children with special reference to home contacts. A tuberculin solution that has been diluted more than two weeks previously should not be used. The whole of the dose (0.1 c.cm.) should be injected intradermally on the outer aspect of the upper arm or thigh. A dilution of 1 in 10,000 should first be used; if that is negative, a dilution of 1 in 1,000 should be given; and if the last is negative a dilution of 1 in 100. For a reaction to be called positive there should be at least 10 mm. erythema and swelling in forty-eight or seventy-two hours. Any reaction below this should be considered doubtful, and the next stronger dilution should be used. In a child under the age of 2 a positive reaction indicates the presence of an active tuberculous lesion unless the contrary is proved by further investigation. In older children a positive reaction merely indicates that tuberculous infection has occurred at some time or other in the past, and that it may or may not be associated with the lesion under suspicion. A negative reaction excludes tuberculous infection provided that consideration is given to the following facts. The reaction may be negative in about 3 per cent of clinically tuberculous persons. Where there is a history of very recent contact, infection cannot be excluded unless the test is repeated at least six weeks after contact has ceased. The following conditions may produce non-reactivity in the presence of tuberculous lesions: very advanced generalized and meningeal tuberculosis, moribund patients, and certain acute infectious diseases. Kayne recommends that all children with a positive Mantoux reaction should have their chests x-rayed.—*Brit. J. Child. Dis.*, January-March, 1936, p. 20. Abs. in *Brit. M. J.*

Men and Books

THE RULES AND REGULATIONS OF AN 18TH CENTURY FRENCH HOSPITAL*

BY T. G. H. DRAKE, M.B., F.R.C.P.(C.),

Toronto

These regulations are contained in a small quarto volume entitled "Compilation of the Ancient Rules made in the general assemblies for the governing of the General Hospital of St. Jacques of Toulon since its foundation, authorized and revised by several general directors' assemblies and especially that of June 6, 1775." This book, published in 1778, is quite rare.

The Hospital received the sick poor, no matter what their country or nationality, suffering from fever, wounds or any other curable malady. Unless coming from the city poor house, it did not admit those suffering from the pest or venereal disease, nor accouchement cases. Patients suffering from scurvy were no longer admitted unless they had a fever. Two wards were devoted to the care of 41 incurable patients. The regulations in many instances are similar to those now adhered to.

"On admission a record is to be made in duplicate by the porter of the family particulars, clothing and moneys of the patient. This is to be checked by the under porter and the money is to be handed over to the treasurer. One copy of this record is attached to the foot of the patient's bed.

"During the day two sisters are to be present in each ward, one of whom shall accompany the physician when he makes his visit to mark down those who are to be placed on special diets.

"Two sisters are to be on night duty. After their supper they are to be instructed by the day sisters of each ward concerning which patients are most in need of care and at what hours nourishment is to be given.

"The patient's diet list is to be attached to the foot of his bed."

Some of the regulations which were necessary in the 18th century, but which do not appear adaptable to present day usage, are the following:

"If the patient dies while in Hospital all that he has brought in with him on admission shall become the property of the Hospital, the clothing to be distributed to needy cases on discharge or sold for the Hospital's benefit.

"No more than one patient shall be placed in each bed, at least not until all the beds are occupied, in which case care shall be taken that a patient suffering from a fever or contagious disease is not placed in the same bed with a convalescent.

"In the evening the pierced chairs shall be placed conveniently at regular intervals. In the morning they shall be removed, except in the cases of those who are unable to go out. The wards shall be aired and fragrant herbs shall be burnt.

"The patients' nightgowns, bonnets and serviettes are to be changed twice a week, more frequently if necessary.

"No food nor drugs shall be given out for use without the Hospital except to those who wish to treat themselves at home for the itch or chlorosis."

Fig. 1.—A Nursing Sister of the 18th Century.

THE MEDICAL AND SURGICAL SERVICES

"The two physicians shall be named by a plurality of votes of the directors after the presentation of dissertations on three subjects which shall be selected by monsieur the treasurer.

"One physician shall visit the male wards, the other the female wards and those suffering from the itch. Their duties shall be alternated yearly. They shall pay two visits each day, at 8 a.m. and 3 p.m. in the winter and at 7 a.m. and 4 p.m. in the summer. They shall sign their orders, which will be written by the surgeon in the register. They shall prescribe the régime which shall be followed in each case. In extraordinary illnesses the physicians shall consult together and, if necessary, with the treasurer's consent, an outside physician may be called in consultation. When a major operation is considered they will consult with the surgeon-in-chief and the attending surgeon, the question of operation being decided by a plurality of votes. When it is necessary to cut out for the stone the physicians will notify the treasurer who will call in one of the lithotomists retained by the city to perform the operation if the physicians consider it necessary.

* From the Department of Pædiatrics, University of Toronto.

"The discharging of all patients is to be under the control of the physicians. In case of necessary absence they shall provide a suitable substitute physician at their own expense. The physicians shall have a voice in all deliberations both public and private. The physician's honorarium shall be at the rate of 500 livres per year, payable six-monthly."

The yearly honorarium mentioned is equivalent to 500 pre-war francs, and the purchasing power of money in 1775 was double what it was in 1913.

"The surgeon-in-chief shall be chosen from the masters of surgery of the city upon the presentation of three subjects decided upon by the treasurer. He shall attend at least twice a week, examining the patients and communicating his knowledge to the attending surgeon.

The surgeon was assisted by two garçons, lodging in the hospital, a position represented by the intern of today, and in addition was allowed to take a certain number of student externs. The garçons were appointed after examination by the physicians and the attending surgeon, the vacancy having been advertised in the school of surgery. They were required to be in for the night before eight in the winter and nine in summer. The garçons were allowed to act only as assistants to the surgeon. They might under his direction do dressings and give enemas in the male wards. Only when the surgeon was unavoidably absent might they care for the female patients, never being stationed

HOTEL-DIEU DE VIENNE EN DAUPHINÉ.

N°.
N°.
N°.
N°.

Je soussigné Curé de Paulhaix en Vivarais certifie avoir vu chez la veuve de Pierre Estrafangle de ma Paroisse l'enfant nommé jean baptiste Badin âgé d'environ douze ans 4 mois [illegible] sans bas, d'ailleurs

enfant dudit Hôtel-Dieu, qui est bien tenu; en foi de quoi j'ai fait le présent.

A Paulhaix ce trentième jour du mois d'octobre 1793 an 2 de la Republique

Fig. 2.—Certificate of the village priest stating that a child placed by a hospital with a wet nurse in his Parish is doing well, 1793.

He shall be present at major operations, but shall never operate without the physician's advice.

"The attending surgeon shall be chosen by majority vote of the directors, physicians, and six masters of surgery after a public dissertation upon a selected subject. If he faithfully serves the hospital for 10 years he shall gain the rights appertaining to a Master of Surgery. He shall be lodged gratis in the Hospital; his salary shall be 120 livres (francs) per year, with 12 measures of wheat at the festival of St. Bartholomew. The attending surgeon shall be given each day for dinner—from which he shall supply his two garçons—2 small loaves of bread and 2 bottles of wine, none of which is to be transported outside the Hospital.

"When the physicians are paying their visits the surgeon shall follow them. He shall appraise them of their previous orders and inscribe in the register any new ones which they shall make. He shall do the dressings and administer the enemas to the male patients, also to the female patients if the sisters are unable to perform these duties themselves.

"The attending surgeon shall shave the priests, officers, domestics and patients twice a week."

for two consecutive weeks on the same female ward.

THE PÆDIATRIC SERVICE

From 1690 until 1728 only foundlings and legitimately born infants of the poor were admitted to the Hospital. In 1728 infants of the poor, born out of wedlock, were admitted directly to the Hospital without the parents having to desert them in the streets to render them eligible for reception. All exposed infants whose parents were unknown were received at the Hospital of St. Jacques, fed on the breast, and later cared for at the expense of St. Jacques until they attained the age of seven years, when they were transferred to the poor house. The poor infants whose known mothers had abandoned them through misery, sickness, or death were cared for by St. Jacques only until the

end of their second year of life, when they were received by the poor house, there to be fed and lodged as were the other poor. All exposed and deserted infants, no matter of what age, who had already been weaned at the time of initial admission were to be sent to the poor house to be treated as mendicants. Any infant's refusal to take the breast was sufficient proof that weaning had taken place.

"For legitimate infants born in the city and suburbs whose parents are unable to care for them, and where the mother has no breast milk to nourish them, application for admission to the Hospital shall be made accompanied by the infant's birth certificate and a certificate of the parents' poverty signed by their priest. After the mother has been visited by the Hospital's physician or surgeon to ascertain that she has no breast milk for the infant's nourishment, and to make sure that she is in reality the infant's mother, the infant is admitted to the Hospital.

"To prevent the exposure of infants, which the midwives are accustomed to practice, the city officials are to be notified of any lying-in-women taken in by the midwives. They will visit the expectant mother and warn the midwife that if the woman is allowed to leave after the confinement without taking the baby with her that the midwife will become liable for the expenses of the infant's care. Infants are not to be placed with wet nurses by midwives or others without the depositor knowing the parents' names and being assured that they are able to pay the nurses, otherwise the depositor herself will become liable for the cost of wet nursing.

"In the Hospital there are to be retained at least two wet nurses, this number to be augmented as necessary, to care for the foundlings or other infants who may be admitted. These wet nurses are hired only after they have been visited by the physicians or, in their absence, by the surgeon and their milk has been found to be good. They will live in their own quarters without any communication with the ill patients. They shall not go outside the Hospital without the permission of the sister superior. They shall wash and dry the infant's clothing. Their wages shall be paid at six-monthly intervals.

"If the available house wet nurses cannot nourish all the infants, they are to be placed out in the country with wet nurses after a medical examination of the nurse by the physician or surgeon. If the milk is found to be good one infant only is confided to each wet nurse, together with the necessary clothing. A billet giving the infant's life and feeding is to be given to the nurse. The country wet nurse is to be paid 4½ livres per month until the infant has attained the age of seven years. No payments are to be made to the wet nurse until they produce a certificate signed by their parish priest stating that the infant is living and doing well.

"One of the directors shall be named in general assembly to care for and dispense the funds allotted for the treatment of infants. He shall see that they are breast fed and pay the salaries of the wet nurses living in the country, upon the certificate of their village priest stating that the infant is alive and well. He shall provide them with the necessary clothing every six months."

THE OBSTETRICAL SERVICE

While St. Jacques did not ordinarily admit women for confinement it did take in these cases when the prospective mother emanated from the city poor house. The city poor house, wishing to be relieved of their responsibilities and costs, transferred many obstetrical cases to St. Jacques much in advance of the time of confinement, and St. Jacques returned those in whom they believed labour was not imminent to the city poor house. To clarify the situation, in 1753 an agreement was ratified between the two disputants that if the patient was sent to St. Jacques more than eight days before her confinement the extra days were to be paid for by the poor house. If the pregnant woman was returned to the poor house before her confinement and the birth took place in less than eight days, the expenses of the confinement were to be borne by St. Jacques.

Hospital Service Department Notes

The Grounding of Anæsthesia Apparatus

Enquiry was made recently concerning the procedure followed in various representative Canadian hospitals with respect to the "grounding" of anæsthesia apparatus.

In the majority of these hospitals the apparatus was not grounded. However, one institution, located where there is considerable static, grounds very carefully; another has orders to that effect, but such are not closely followed; one eastern hospital grounds only when cyclopropane or gas-oxygen-ether mixtures are being used in cold weather; one hospital, following an undesirable incident, has decided to "ground everything". In one Pacific coast hospital it is considered that the natural humidity of the coastal regions makes the danger from static electricity practically negligible. Some anæsthetists are of the opinion that, with the rebreathing technique, the danger of explosion is greatly minimized.

There would seem to be general agreement concerning the necessity of taking all reasonable precautions against explosion, but opinion does not seem to be settled as to whether or not grounding helps to minimize the danger. Might it not add to the hazard? Dr. Ralph Waters of Madison, Wisconsin, has stated, "Attempts to ground certain objects, such as the anæsthetic machine, in the presence of failure to ground other objects, such as patients wheeled in on a rubber tired cart, add to the likelihood of differences in electric potential. It is therefore recommended that no attempt to ground any object in the operating room be made." The National Board of Fire Underwriters (N.Y.C.) in its booklet "Fire Prevention and Protection as Applied to Hospitals," 1929, did recommend the bonding together of the patient, the table,

All communications intended for the Department of Hospital Service of the Canadian Medical Association should be addressed to Dr. Harvey Agnew, 184 College Street, Toronto.

the surgeon, the anæsthetist and the anæsthetic apparatus, all to be thoroughly grounded, but the following year a special committee of the American Medical Association doubted whether grounding would give more than an incomplete protection, "for the most serious explosions are probably initiated by electrical discharges of static electricity developed inside the anæsthetic apparatus itself. Grounding considerably increases the danger of a short circuit from the electrical illuminating current to the patient, surgeon and anæsthetist."

An excellent article on "Safeguarding the operating-room against explosions" appeared in the April, 1935, issue of *Modern Hospital* and was followed in the May number by a valuable "Code of safeguards". The author, Mr. Victor B. Phillips, of the University Hospitals, Cleveland, concluded that "the present evidence on the whole is against the grounding method, whether or not operating-room atmosphere is adequately humidified, but it must be made clear that in the absence of proper room humidity the lack of grounding leaves no protection against static developed outside of the anæsthesia machine. It means the acceptance of one hazard as being on the whole a lesser evil than the other hazards which grounding induces."

There does seem to be agreement, however, upon the value of proper air conditioning, particularly with respect to humidification and ventilation. One large eastern hospital recommends a humidity of over 50 per cent. Mr. Phillips suggests an R.H. of between 58 and 63 per cent in the rooms and not less than 50 per cent in the adjacent corridors. Dr. Waters stated recently, "Operating rooms with a relative humidity exceeding 60 per cent are probably safe from the static hazard".

Other precautions were suggested. In one hospital all entering the operating-room must wear leather-soled shoes; rubber-soled shoes are not permitted. All electrical connections should be checked, and any switch or adjustable transformer likely to produce sparking should be replaced. The danger of short-circuiting, particularly with grounded objects, warrants very careful attention to insulation. In one hospital the last duty at night is to inspect the anæsthetic machines and remove the plugs from the wall. Movement around the patient prior to the anæsthetic should be minimized. Static associated with persons entering the operating-room should be discharged by touching a grounded plate at the entrance. Wool blankets should not be used in the operating room. Open flames, cauteries, cigarettes, etc., create a dangerous hazard in the presence of inflammable or explosive gases.

Provincial Association Notes

The Manitoba Medical Association

The Annual Meeting of the Manitoba Medical Association was held in the Fort Garry Hotel, Winnipeg, May 14th, 15th and 16th. On the opening day there was a skin clinic at which cases were presented by Drs. A. M. Davidson and G. B. Bedford. Dr. Stewart Schultz, of the Brandon Mental Hospital, who has recently been awarded the Prowse prize, gave an interesting address on "Industry as applied to treatment in mental hospitals". Then followed a clinical pathological conference presided over by Professors C. R. Gilmour and Wm. Boyd.

The afternoon was given over to golf at the St. Charles Country Club. The winner of the Cup was Dr. S. Bardal, of Shoal Lake, and the runner-up, Dr. E. S. Moorhead, of Winnipeg.

In the evening Dr. Alexander Gibson gave a talk on "The treatment of knee-joint injuries". Dr. Charles Hunter pointed out "Common errors in medical practice". The most frequent error, he said, was the failure to obtain a history. In many instances a carefully taken history would obviate the necessity of special examinations. Another common error was failure to make a complete physical examination. Rectal and pelvic examinations, as well as examinations of eye grounds, were often neglected.

Dr. E. W. Montgomery spoke on "Tuberculosis as a public health problem". The particular obstacle in the lowering of the tuberculosis death rate in Manitoba, he said, was due to the very widespread incidence of the disease among the 15,000 Indians of Manitoba. Forming less than 2 per cent of the population they contributed 28 per cent of the deaths from tuberculosis and were a constant course of infection. The Indians are wards of the Federal Government, but unless some action is taken towards combating the disease among them there is little chance of preventing constant infection from this source. All three papers were well discussed.

The morning of May 15th was given up to surgical clinics at the Winnipeg General Hospital under the direction of Dr. C. W. Burns. The first hour was given to the "Tumour Clinic", when a number of cancer cases, chiefly gynæcological, were presented. Following this those in attendance divided into two groups, Section A being under the chairmanship of Dr. G. S. Fahrni and Section B under Dr. P. H. T. Thorlakson.

The annual general meeting was held in the Fort Garry Hotel. Dr. F. G. McGuinness, the retiring President, gave an address on "The medical association as an educational unit". The meeting stood for a minute's silence in honour of Dr. W. H. Secord, a

former member of the Manitoba Medical Association Executive, Dr. T. H. Bell and Dr. J. T. Wright. Dr. C. W. Burns, the Treasurer, submitted an excellent report showing a considerable balance on hand. The election of officers resulted as follows: *President*, Dr. George Clingan, Virden; *First Vice-president*, Dr. J. D. Adamson; *Second Vice-president*, Dr. P. H. T. Thorlakson; *Treasurer*, Dr. C. W. Burns; *Secretary*, Dr. F. W. Jackson; *Rural Members at Large*, Drs. J. K. Cunningham, Carman, and S. Bardal, Shoal Lake; *Winnipeg Member at Large*, Dr. S. G. Herbert.

Reports were presented from the Executive Committee by the Committee on Sociology, the Committee on Historical Medicine and Necrology, the Legislative Committee, the Radio Committee, the Committee on Maternal Mortality, the Editorial Committee, the Editorial Board of the *Canadian Medical Association Journal*, the Extra-mural Committee, the Workmen's Compensation Referee Board, and the Committee on Federation Proposal.

The recommendation of the last mentioned committee, which was passed as a resolution by the general meeting, was as follows: "It is the view of your committee that cooperation of the provincial medical associations through a central body, according to the principles suggested in the Manitoba Medical Association memorandum of October, 1934, is still desirable, but that these principles are not applied in the status of a 'division' of the Canadian Medical Association under the 'Federation proposal'. Your committee therefore recommends that action on the 'federation proposal' be deferred."

Dean A. T. Mathers introduced to the meeting Dr. John Kilgour, a 1936 graduate of Medicine, University of Manitoba, who was awarded the Gold Medal of the Manitoba Medical Association for the highest standing in the five years of the medical course. He is a nephew of the late Dr. Thomas McCrae, and Lieut.-Col. John McCrae.

Following the annual meeting a scientific meeting was held under the chairmanship of Professor C. R. Gilmour. Dr. L. G. Bell spoke on "Convulsions in hypertensive states"; Dr. Charles Walton on "Hypoglycæmia"; Dr. Gilbert Adamson on "Nervous symptoms and disturbances of the menstrual cycle"; Dr. A. Hollenberg on "Kidney conditions complicating pregnancy", and Dr. George Black on "Treatment of syphilis".

The annual dinner and dance was held on Friday evening in the Fort Garry.

On May 16th Dr. Brian Best, of Killarney, read an interesting paper on "Golden rules in obstetrics". Dr. C. K. Cunningham, of Carman, spoke on the treatment of fractures from the standpoint of the general practitioner. Dr. T. E. Holland discussed "The injection treatment of hernia", and Dr. Oliver Waugh, "The treatment of trigeminal neuralgia".

Dr. F. G. McGuinness was the host at a dinner to the retiring executive at the Manitoba Club on May 13th at which the Hon. I. B. Griffiths, Minister of Health and Public Welfare, and John Queen, Mayor of Winnipeg, were present.

The ladies' program comprised an afternoon tea given by Mrs. F. G. McGuinness at her residence, 61 Cordova Street, an afternoon tea on May 14th and a luncheon on May 15th at the Manitoba Club to the wives of the retiring executive given by Mrs. McGuinness.

ROSS MITCHELL

Medical Societies

The Colchester-East Hants Medical Society

This Society met at Truro and elected its officers for the coming year: *President*, Dr. Dan Murray, Tatamagouche; *Vice-president*, Dr. H. B. Havey, Stewiacke; *Secretary-treasurer*, Dr. D. S. McCurdy; *Executive to the N. S. Medical Society*, Drs. H. R. Peel and D. F. McInnes.

Montreal Dermatological Society

This Society meets thrice a year, the annual meeting being held in October. *President*, Dr. B. Usher; *Secretary-Treasurer*, Dr. Paul Poirier.

The Saint John Medical Society

The annual meeting of the Saint John Medical Society was held at the Ridgewood Golf and Country Club on May 27. Dr. W. J. Baxter, Fairville, retiring president, in his parting address, suggested a revision of the by-laws of the Society and made other recommendations to increase the interest in the Society over which he has presided most capably during the past year. Following the annual dinner Dr. A. E. Macaulay exhibited a series of moving pictures, which included a record of his visit to the Old Country during the recent Jubilee year.

Election of officers for the coming year resulted in the following slate being appointed: *President*, Dr. F. C. Jennings; *Vice-president*, Dr. C. O. MacKay; *Secretary*, Dr. V. A. Snow, Hampton, N.B.; *Treasurer*, Dr. G. B. Peat (reappointed); *Executive Members*, Drs. M. McDonald, H. A. Farris, and Joseph Tanzman.

The Valley Medical Society

In annual conclave, the Valley Medical Society has elected the following officers: *President*, Dr. O. R. Stone, Bridgetown; *Vice-presidents*, Drs. J. R. McCleave, Digby, J. R. Sutherland, Annapolis, and T. A. Kirkpatrick, Kentville;

Secretary-treasurer, Dr. H. E. Kelly; *Executive to the N. S. Medical Society,* Drs. A. B. Campbell and V. D. Schaffner.

The Western Nova Scotia Medical Society

At its annual meeting the Western Nova Scotia Medical Society elected the following officers: *President,* Dr. C. K. Fuller; *Vice-Presidents,* Drs. Z. Hawkins and H. H. Banks; *Secretary-treasurer,* Dr. T. A. Lebbetter; *Executive to the Nova Scotia Medical Society,* Drs. V. Burton and J. E. LeBlanc.

The Winnipeg Medical Society

At the annual meeting of the Winnipeg Medical Society held on May 8th the following officers were elected for the ensuing year: *President,* Dr. W. E. Campbell; *Vice-president,* Dr. G. E. Corrigan; *Secretary,* Dr. R. W. Richardson; *Treasurer,* Dr. K. C. McGibbon; *Trustees,* Drs. A. Kobrinsky, A. W. Hogg, and S. E. Elkin.

ROSS MITCHELL

Post-Graduate Courses

Queen's University

The Medical Faculty of Queen's University, Kingston, announces a post-graduate course to be held from September 7 to 11, 1936. The program will cover medicine, surgery, gynæcology, bacteriology, radiology, and special apparatus, and practitioners may attend all or part of the course. Registration fee is $2.00.

Full details may be obtained from Dr. L. J. Austin, Chairman of Arrangements, Kingston General Hospital.

The University of Toronto

POST-GRADUATE COURSE IN ABDOMINAL SURGERY

The University of Toronto, Faculty of Medicine, offers to graduates in Medicine who have had a satisfactory training in Surgery, a Course of one week, commencing Monday, September 28th, on the General Surgery of the Abdomen.

This Course will consist of lectures, demonstrations, clinics, operations and practical instruction on the cadaver. It will be conducted at the Toronto General Hospital, St. Michael's Hospital, The Hospital for Sick Children, the Toronto Western Hospital, and the Banting Institute. It will commence at 11 a.m. on Monday, September 28th, at the Toronto General Hospital and will continue, morning and afternoon, until Saturday, October 3rd at noon.

The subjects for discussion and demonstration will be as follows:

Abdominal wall.—Anatomy, incisions, closure, hernia.

Small intestine.—Obstruction, intestinal suture, resection, ileostomy.

Large intestine. — Circulation, radiological study, obstruction, intussusception, resection for carcinoma of cæcum, hepatic flexure, transverse colon, splenic flexure, descending colon and sigmoid, Mikulicz operation, cæcostomy, colostomy, care of permanent colostomy, Hirschsprung's disease.

Rectum. — Examination, use of proctoscope and sigmoidoscope, treatment of hæmorrhoids; carcinoma, abdomino-perineal and perineal operations for cure.

Gall-bladder and bile ducts.—Anatomy, cholecystitis and gall-stones, cholecystectomy, obstruction of common duct, etc.

Stomach and duodenum. — Anatomy, pyloric stenosis, gastric and duodenal ulcer, gastric carcinoma, radiological study, gastroenterostomy, pyloroplasty, gastrectomy.

Spleen.—Banti's disease, hæmolytic icterus, purpura hæmorrhagica, splenectomy.

Appendix.—

The Course will be made as practical as possible, and the students will take part in the demonstrations and operations. An opportunity will be afforded to all to perform the operations on the cadaver.

During this week the San Carlo Opera Company will be singing at Massey Hall, the Promenade Symphony Orchestra will play at the Varsity Arena, and the horse races will be on at the Woodbine.

The Course will be limited to twenty students and to students who have had sufficient practical and theoretical training in surgery to make it possible for them to profit from a rapid and intensive review of the subject, such as this will be.

Applications for admission to the Course should be made to the Secretary, Faculty of Medicine, University of Toronto, before September 1st. With the application must come a statement of the applicant's previous training and experience in abdominal surgery. A fee of $25.00 will be charged, which as usual will be spent on the Course.

The Vancouver Medical Association

A Summer School is held annually under the auspices of the Vancouver Medical Association. The speakers for the 1936 session, which is scheduled for September 8, 9, 10, and 11 inclusive are: Dr. Gordon B. New, Professor of Oto-Laryngology and Rhinology, Department Oral and Plastic Surgery, Mayo Clinic, Rochester, Minn.; Dr. W. McK. Marriott, Professor of Pædiatrics, Department of Pædiatrics, Washington University, St. Louis, Mo.; Dr. Evarts A. Graham, Professor of Surgery,

Department of Surgery, Washington University, St. Louis, Mo.; Dr. J. McF. Bergland, Lecturer in Clinical Obstetrics, Baltimore, Md., Johns Hopkins University School of Medicine; Dr. C. B. Farrar, Director, Toronto Psychiatric Hospital, Toronto; Dr. Rollin T. Woodyatt, Clinical Professor of Medicine, Chicago, Ill., Rush Medical College, University of Chicago; Dr. C. E. Dolman, Director of the Provincial Health Laboratory, Vancouver, B.C.

H. A. DESBRISAY,
Secretary Summer School Committee.

University Notes

The University of Western Ontario Medical School

ANNUAL REFRESHER COURSE

The University of Western Ontario wishes to direct attention to the Refresher Course which will be given next September as follows:

Monday, September 14th, 9 a.m. to 1 p.m.—Surgery.

Tuesday, September 15th, 9 a.m. to 1 p.m.—Medicine.

Wednesday, September 16, 9 a.m. to 12 p.m.—Obstetrics and Gynæcology. 2 p.m. to 4 p.m.—Clinical Laboratory Procedures and Pathological Demonstrations.

Thursday, September 17th, 9 a.m. to 12 p.m.—Pædiatrics.

Friday, September 18th, 9 a.m. to 1 p.m.—Eye, Ear, Nose and Throat Radiology.

Detailed programs will be mailed upon request to all who are interested. Address inquiries to: The Dean, University of Western Ontario Medical School, London.

Dalhousie University

COURSE IN PUBLIC ADMINISTRATION

Dalhousie University announces a new Honours course in Public Administration to begin September, 1936. This course is designed particularly for students looking forward to a career in the Civil Service (Federal, Provincial or Municipal). It should also be of great value to students looking forward to such professions as Law or Journalism, or other careers having to do with public affairs. Its aim is to provide the necessary background rather than a technical training.

The need for special training for candidates for the public service is apparent. Governments are being compelled, very often against their wishes, to take on many new responsibilities, especially in the economic and social spheres. To carry out its many tasks efficiently a government requires an administrative staff of high ability and sound education, otherwise inefficiency and waste are likely to add to the costs. Special training for prospective civil servants has long been provided by the University of London. More recently several other universities throughout the British Empire have begun to provide this training, as have also a few American universities, notably Harvard and Chicago. Hitherto no Canadian university has undertaken work in this field. Dalhousie is, therefore, a pioneer in offering this course, as it was when it established the first academic law school in Canada more than half a century ago.

Dalhousie University is peculiarly well equipped to conduct such a course, in that it not only has a well-staffed Faculty of Arts, but can also draw on the Faculties of Law and Medicine. For this course the teaching resources of departments in all three faculties will be pooled. While the work naturally falls under the direction of the department of Political Science, classes will be included from the related fields of History, Economics, Law, Commerce, Public Health, and there will be special classes in Public Administration. These classes will be for general students, as well as for those taking the Honours course.

It is also hoped that facilities will be provided for special investigations and researches in public administration, in subjects of particular interest to Nova Scotia, the Maritime Provinces and to Canada as a whole.

Dalhousie University conferred medical degrees on twenty-six of the twenty-nine candidates who wrote the final examinations. Dr. Fraser Young, of Pictou, a son of Dr. M. R. Young (Queen's '01), won the rarely awarded Gold Medal.

McGill University

At the Convocation held on May 28, 1936, the degree of M.D., C.M. was conferred on ninety-two candidates. For the first time in many years there were no failures in the final examinations, and, to signalize the occasion, the graduating class presented Dean Martin with a pair of white gloves!

The prize winners were the following: The Holmes Gold Medal for the highest aggregate in all subjects forming the Medical curriculum; second place aggregate standing in the final year. —Ebert E. Judd, B.A., Waltham, Que. The Wood Gold Medal for the best Clinical Examinations in the subjects of the final year; seventh place aggregate standing in the final year.—David W. MacKenzie, B.A., Montreal. The Lieutenant-Governor's Silver Medal for the highest standing in Public Health and Preventive Medicine; The Jeanie Forsyth Prize for high standing in Surgery; first place aggregate standing in the final year. — Harold Rocke Robertson, Vancouver, B.C. The Alexander D. Stewart Prize for the highest general qualifica-

tions for the Practice of Medicine; sixth place aggregate standing in the final year.—Robert G. Freeman, B.A., Pasadena, Cal. The J. Francis Williams Fellowship in Medicine and Clinical Medicine; fourth place aggregate standing in the final year.—Rachmiel Levine, B.A., Outremont, Que. Diplomas in Public Health were granted to: John Merrill Cruikshank, M.D., C.M. (McGill), Nassau, Bahamas; John Sutherland Douglas, M.D. (Toronto), Ottawa, Ont.

Dr. C. F. Martin, retiring Dean of the Medical Faculty was granted the degree of Doctor of Laws, *honoris causa.*

The University of Manitoba

At the recent convocation the following medals and prizes in the Faculty of Medicine were awarded: The University Gold Medal.—John McCrae Kilgour, B.Sc. Manitoba Medical Association Gold Medal (for highest standing on the first four years of the course).—John McCrae Kilgour, B.Sc. Dr. Charlotte W. Ross Gold Medal (for highest standing in obstetrics in the course).—Nathan Goluboff. Chown Prize in Medicine, gold medal and $50.00.—John McCrae Gilgour, B.Sc. Chown Prize in Surgery, gold medal and $50.00. — Frederick Robt. Tucker. Prowse Prize for Clinical Research in Medicine: For Research on the Rôle of Industry in Psychiatry.—Stewart Duncan Schultz, B.A., M.D., Brandon Mental Hospital, $250.00. The Physiological Research Prize (for an original investigation carried out wholly or in part in the Department of Physiology or the Department of Bio-chemistry of the University of Manitoba)—Bronze medal and $10.00.—Abram Neufeld, M.Sc., for "Contributions to the Bio-Chemistry of Bromine." Bronze medal and $10.00.—William Donald Ross, for "The Innervation of the Thyroid Gland." Isbister Scholarships were awarded as follows: First year, first scholarship, $100.—Beatty H. Ramsay; second scholarship, $80.—Eric F. Chapman. Second year, first scholarship, $100.—J. Edward Musgrove; second scholarship, $80.—Edward A. Sellers. Third year, first scholarship, $100.—Arthur Lerner; second scholarship, $80.—Alexander M. Ogryzlo. Fourth year, first scholarship, $100.—Gerda Fremming; second scholarship, $80.—Robt. M. Ramsay.

At a meeting of the Medical Faculty Council held on June 2nd the following members of the Executive were appointed: Dean A. T. Mathers, Professors C. R. Gilmour and John Gunn, ex-officio. Dr. F. G. MacGuinness, Prof. Wm. Boyd, Prof. A. T. Cameron, Dr. D. S. MacKay, Dr. A. P. MacKinnon and Prof. R. G. Inkster. Prof. R. G. Inkster and Dr. Ross Mitchell were elected representatives of the Medical Faculty to the Senate of the University.

Oxford University

An anonymous donor has given £10,000 for the establishment of an Institute of Experimental Psychology at Oxford University. It is proposed to allocate to the Institute £500 and £150 per annum from the Rockefeller Foundation for research in social sciences.

University of Toronto

Medals, prizes, scholarships and fellowships awarded by the Senate of the University Faculty of Medicine, 1936.

Sixth Year.—The Faculty Gold Medal.—O. M. Solandt, B.Sc.(Med.), M.A. The Faculty Silver Medal.—C. H. Jaimet. The Faculty Silver Medal.—A. M. Large, B.A. The Ellen Mickle Fellowship, O. M. Solandt. The Chappell Prize in Clinical Medicine.—O. M. Solandt. The William John Hendry Memorial Scholarship in Obstetrics and Gynæcology.—O. M. Solandt. The Ontario Medical Association Prize in Preventive Medicine.—O. M. Solandt. The David Dunlap Memorial Scholarship.—O. M. Solandt. The Canadian Medical Institute Prize.—O. M. Solandt.

Undergraduate.—The John Copp Bursary.—C. C. Gray. The David Dunlap Memorial Scholarships.—(a) Fifth Year, W. R. Feasby, B.A.; (b) Third Year, J. C. McCulloch. The Ronald S. Saddington Medal in Pathology.—E. B. Tovee. The Baptie Scholarship.—J. H. Tritt.

Graduate.—The Reeve Prize.—M. H. Roepke, B.S., M.S., M.A., Ph.D., and S. L. Cohen, B.A. The George Brown Memorial Scholarship in Medical Sciences. — R. E. Haist, B.A. The Alexander McPhedran Research Fellowship in Clinical Medicine.—R. C. Dickson, M.D. The Perry Goldsmith Prize in Oto-Laryngology.—J. A. Sullivan, M.B. The J. J. Mackenzie Fellowship in Pathology and Bacteriology.—R. P. Douglas, M.D. The Lister Prize in Surgery.—H. R. Ziegler, M.D.

Cambridge University

Being greatly impressed by the value of the work of Lord Rutherford in the realm of scientific research Sir Herbert Austin has presented to Cambridge University securities to the value of approximately £250,000. Lord Rutherford states that the donation will afford an opportunity of building a modern research laboratory and will also be of great value in helping to defray the great expense attached to the prosecution of modern research in physics. The first use made of the money will be to build a laboratory for the utilization of very high voltages in order to carry out experiments on the transmutation of matter by high-speed particles and by radiation.

Special Correspondence

The Edinburgh Letter

(From our own correspondent)

The Committee appointed in June, 1933, by the Secretary of State for Scotland "to review the existing health services of Scotland in the light of modern conditions and knowledge, and to make recommendations on any changes in policy and organization that may be considered necessary for the promotion of efficiency and economy" has now completed its remit. The Committee suffered a severe loss by the death of the Chairman, the Hon. Sir John C. Dove Wilson, after it had been sitting for a year and a half. Prof. E. P. Cathcart, of Glasgow University, a member of the Committee, was appointed to succeed Sir John as Chairman. The Committee has had an enormous volume of evidence presented to it. Part One of the Report begins with a brief account of the growth of the health services, to bring out the expanding conception of public health, proceeds to a summary review of the evidence received on population growth and distribution, social and economic conditions, and the state of health of the people, and concludes with a statement of the conclusions regarding the general lines along which, in its view, health policy should now be directed. From that point the Committee was able to review the existing services and to consider which changes in policy and organization were necessary "for the promotion of efficiency and economy". Part Two deals with Heredity, Nutrition, Education and the Environmental services; Part Three, with the Medical and Allied services; Part Four, with Finance and Administration; and Part Five presents a General Conclusion and Summary. In framing its proposals the Committee has endeavoured to view the problem of National Health as a whole, and to provide for a flexible structure, so that, at any time and according to circumstances, effort may be directed where it would yield the best results. The opinion is expressed that, within limits, health may be purchased, and the Committee is satisfied that with expenditure in the application of established knowledge, and with the cooperation of the people themselves, the general standard of health in Scotland could be raised to a higher level. The standard at present is far below what can be attained. The Committee's proposals are submitted in the belief that the additional expenditure involved would yield a good return in individual happiness and in national efficiency. The Report is expected to be available to the public towards the end of June. It will be studied with the greatest interest by the medical profession, those responsible for the administration of the health services, and by a large section of the general public. In future letters I hope to deal specifically with some of the more important recommendations made in the Report.

The news of the sudden death of Field-Marshall Viscount Allenby at his home in Kensington, London, was received with great regret in Edinburgh. Little more than a fortnight previously he had spent a week in the City and had delivered his Rectorial address to the students of the University. The proceedings at the address were characterized by frequent interruptions by a noisy and irresponsible section of the audience, and this matter has been the subject of a considerable amount of correspondence in the local press. The matter is now receiving the attention of the University authorities. In his address Lord Allenby denounced the "barren business of war" and appealed particularly to the young to help to attain world peace by international agreement.

The appointment of Sir Hector James Wright-Hetherington, M.A., LL.D., Vice-Chancellor of Liverpool University, to be Principal of Glasgow University in succession to Sir Robert Sangster Rait, C.B.E., M.A., LL.D., whose resignation is to take place on September 30th, has been approved by the King. Sir Hector, who is 48 years of age, had a distinguished undergraduate career in Arts at Glasgow University. In 1924 he was appointed to the Chair of Moral Philosophy at that University, a post which he held until his appointment as Vice-Chancellor of the University of Liverpool in 1927. He has been a member of various important Government bodies, including the Royal Commission on Unemployment Insurance. In 1935 he acted as Chairman of the Board of Inquiry into the wages agreement in the cotton manufacturing industry.

At a luncheon held recently to mark the close of the sixth year in the life of the Cosmopolitan Club of Edinburgh University over ten different nationalities were represented. Among those present were students from Germany, India, the West Indies, West Africa, China, Switzerland, Mauritius, Russia, and South Africa. Mr. Robert S. Hoffman, of the United States, presided at the luncheon. Professor Sir David Wilkie, one of the honorary presidents of the Club, was the principal speaker. He said that the future peace of the world depended very largely on those who were now studying in Universities in this and in other countries, and such a Club provided an international link which might do much to improve the present relationship between the nations of the world.

At a recent meeting of the University Court of Edinburgh it was announced that a grant had been received from the Rockefeller Foundation of £7,500 for research work in Neuro-Surgery during the five years from June 1, 1936. The work is to be carried out under

the direction of Mr. Norman Dott, F.R.C.S.E., of Edinburgh, whose work in this field is so well known.

R. W. CRAIG.

7 Drumsheugh Gardens,
Edinburgh.

The London Letter

(From our own correspondent)

Thirteen years ago the grading of milk was introduced as a contribution towards solving the problem of a clean milk supply. As events have turned out the main benefit of such a scheme has been to the producer rather than to the consumer. Just as this letter leaves a new scheme of grading will come into force which does little to improve the position and nothing towards the provision of that "safe" milk supply which has been the demand of enlightened workers for the health of the nation for some years past. Sir Gowland Hopkins presided over a committee for two years which in 1934 produced an excellent report indicating how our milk supply could be regulated on scientific lines. No attempt has been made to implement this report and the profession must feel profound disappointment with the new regulations. One well-known medical officer of health refers openly to some of the milk sold in his area as "liquid dung" but he is powerless to prevent it and recently one enterprising milk producer actually did add some manure to a specimen submitted to the authorities, only to receive an assurance that the standard of this milk was being well maintained.

These are ugly facts but they cannot be denied and although peaceful methods of attempting to improve the milk supply of the nation seem to have failed there may be still a faint hope in some war-time scheme! This is not to suggest that war is yet a certainty, but undoubtedly recent international events have led to a close study of the question of food supply in time of war and it is even rumored that a "food controller" or some such dictator has been appointed. An interesting leading article in the *British Medical Journal* dealt recently with this problem, urging the scientific storage of a certain amount of food-stuff as an emergency ration, so to speak. This was to include hard whole wheat from Canada or Australia, potatoes, onions, cheese, butter, sugar and dried, whole or skimmed milk. Could the nation be made self-supporting as regards milk, eggs, vegetables, oats, barley and meat this ration could be modified, and the article goes on to urge the great importance of becoming self-supporting in milk and milk-products from several points of view. Only a dictator can possibly force all the parties concerned to produce a safe milk.

The cynic might well point out that the new poisons' rules come into force just one month before the new milk regulations. Perhaps if two separate government departments had not been concerned the two orders might have been usefully combined. Certainly the present lists of poisons for which special signatures and prescriptions are required contain some dangerous substances but there are also others little more dangerous than ordinary milk! Under the new poisons rules everyone concerned with drugs has different duties—except the patient. The pharmacist is mostly affected, but the medical practitioner, the hospital, the dentist and the veterinary surgeon will be compelled to use new methods in buying poisons and prescribing them. As is so often the case nowadays, new laws are more complicated than the old ones they replace. Thus the schedules of poisons under the new Act would occupy many pages of the ordinary book and the list of substances to which special restrictions apply contains the names of over a thousand articles. It is not surprising that many hospitals have decided that in future all medicines prescribed for in- or out-patients must be signed for by the practitioner concerned. What this will mean in a busy out-patient department can readily be imagined. Writer's cramp among out-patient physicians will probably become quite common!

Surgical research has recently received yet another big stimulus in the shape of a gift of £25,000 from the Bernward Baron trustees. It is a little over five years ago that facilities were provided for research of a surgical and pathological character at the College of Surgeons in London and shortly after this Sir Buckstone Browne endowed the research farm at Downe with a residential hostel attached. Already young surgeons have made important contributions from these places on such subjects as "shock" and "intestinal obstruction". The new gift will enable the College to build and equip new laboratories where surgeons already on the active staff of the various hospitals can find facilities for aspects of research suggested by their everyday clinical work.

121 Harley St., ALAN MONCRIEFF.
London, W.1.

LIBRARIANS HAD TROUBLES, EVEN IN ANCIENT GREECE.—Even in cultured old Athens, it turns out, Greek readers would sneak books out of libraries, against the rules. A marble sign that some desperate librarian set up, 1,800 years ago, has been unearthed by American excavators in Athens: "No book shall be taken out. We have sworn it! The library will be open from the first hour until the sixth." The notice, inscribed in Pentelic marble, has been uncovered by the expedition of the American School of Classical Studies. The Greek inscription was dug up in the Athenian market place, and is believed to have belonged in front of the Library of Trajan, about 100 A.D. Library hours, "first hour until the sixth," were from seven in the morning until noon. —*Science News Letter*, March 28, 1936.

Topics of Current Interest

Scientific Discoveries and Patents

The problem of medical patents has been agitating the medical profession increasingly in recent years. The "Principles of Medical Ethics" says quite plainly "It is unprofessional to receive remuneration from patents for surgical instruments or medicines; to accept rebates on prescriptions or surgical appliances, or perquisites from attendants who aid in the care of patients."

Through the centuries, medicine has given freely of its discoveries for the benefit of mankind. Vaccination against smallpox, inoculation against hydrophobia, digitalis, and innumerable other methods and medicaments became the property of all who cared to employ them in the control of disease. Now, as medicine has become more complex, involving technical assistants in the fields of biochemistry, physiology, physics and associated branches, great numbers of people who give their full time to the work of the hospital, the laboratory or the care of the sick work with the medical profession but are not bound by the same ethical principles. Moreover, the universities have developed preparations and techniques in their laboratories at considerable expense to the institution, and workers have seen fit to turn over to the universities the control of such products. Thus insulin is controlled by the governors of the University of Toronto; scarlet fever preparations are controlled by the Scarlet Fever Committee, Inc.; vitamin D preparations by the Wisconsin Alumni Research Foundation of the University of Wisconsin; the Sperti patents by the University of Cincinnati; the Zucker patents by Columbia University; the Doisy patents are controlled by the St. Louis University School of Medicine, and several patents are the property of Stanford University and the University of California.

The American Association for the Advancement of Science published in 1934 a report on this subject with the arguments for and against commercialization of university patents. The problem continues to be studied by the National Research Council and by many other bodies. The question is one that has concerned the House of Delegates of the American Medical Association, and the Board of Trustees even now is engaged in a survey of the situation.

In the current issue of *Harper's Magazine*, George W. Gray* presents an interesting analysis of the present status of affairs as it concerns problems not only in the medical field but in science generally. He points out that separate patent-holding agencies were operating at the beginning of 1936 at the Universities of Cincinnati, Columbia, Cornell, Iowa State College, Lehigh, Pennsylvania State College, Purdue, Rutgers, Utah, Wisconsin, and Wittenberg College. Moreover, the state universities of Illinois and Minnesota, the California and Massachusetts Institutes of Technology, and the Franklin Institute of Philadelphia control either directly or by a committee patents contributed by research workers.

There seem to be three contrasting methods of financial control of university patents: (1) the method of the general holding company; (2) the method of the university holding company; and (3) the direct or committee method. Apparently these methods were established with the chief objective of protecting the public against substitutes and makeshifts. By means of the patent the inventor can insure that whatever is offered to the public under his invention is technically right.

Mr. Gray feels that, when a new device involves the purity of a process or of ingredients, the public interest is best served by patenting. However, service of the public interest by insuring purity and quality of preparations is far different from commercial exploitation of the fruits of university research.

One of the most cogent arguments now advanced concerns the relationship of a university or research institution to taxation. Endowed universities and research institutions are usually exempt from taxation. They are non-profit institutions. As an endowed institution, free from taxation, such institutions owe a definite duty to the public, which bears the burden of taxation. This is certainly a practical consideration which may well engage the authorities in the universities responsible for administration of commercially valuable patents.

Again, the exploitation of patents by various universities places them in direct competition with one another, as, for example, the three patents concerned with the development of vitamin D. Under such circumstances the results of current research are jealously guarded and all research probably delayed through failure of research workers to communicate with one another. Indeed, even workers in the same university develop a competitive spirit likely to destroy entirely the type of cooperation in science which is responsible for much of our current progress. This conception of scientific research is beautifully expressed by Mr. Gray in the following paragraph:

> Scientific research, as a recognized full-time occupation, is one of the youngest of the professions. It has come up out of the basements and garrets of the early experimenters, and has attained status among the most honoured of the callings of men. Perhaps the laboratory is pressed with economic necessity—but is that warrant

* Gray, G. W.: Science and profits, *Harper's Magazine*, 1936, **172:** 539.

for changing its charter? Possibly it can support itself handsomely and independently—but can it survive the shiftings of bases and the readjustments of outlook which commercialization entails? One of its greatest glories is its intellectual integrity and independence—but can this reputation continue unsullied in the clash of competitive sales campaigns of patented commodities, infringement suits, and other contentions of the marketplace in which the financial interest of the research institution is on one side of the dispute?

A final consideration concerns the rights of the physician. When our civilization was such as to safeguard the physician, considering him as one who gave freely of his knowledge and service to mankind and therefore entitled to special consideration, physicians might well offer freely their time, their service, their original contributions. Our complex civilization of today places ever new burdens on a willing bearer of mankind's tribulations. Surely there was never a problem more complex offered for solution by the best minds of the medical profession.—*J. Am. M. Ass.*, 1936, **106**: 1388.

Medico-Legal

XXI.

Alberta Dental Association v. Sharp[1]

Alberta—Unprofessional conduct of a dentist—Advertising.

Section 31 of the Alberta Dental Association Act provides for the investigation of unbecoming or unprofessional conduct on the part of a licensed dentist by a committee, which if it considers the charge proved is to refer the matter to a District Court Judge who has the power to suspend or cancel the dentist's license. By an amendment to the Act passed in 1927[2] it is provided that, "No person shall advertise or hold himself out to the public as a specialist or as being specially qualified in any particular branch of dentistry or dental work, without having received from the Registrar of the University of Alberta a certificate of having complied with such conditions precedent as to qualification or fitness as may be prescribed by the Senate of the said University". The section further provides that any breach of it is deemed to be unbecoming or unprofessional conduct within the meaning of section 31.

The respondent was a licensed dentist and complaints had been taken to some of his advertising. One of his advertisements appearing in the Edmonton Telephone Directory contained, after the dentist's name, the words "Specializing in Crown and Bridge Work and Dental Plates. Have you seen my Hecolite Plates?" Some of the other advertisements complained of contained prices for various classes of work, many of them with illustrations, and some of them comparing his prices with those charged elsewhere. Proceedings were taken under Section 31, and the District Court Judge who heard the case found him guilty of unprofessional conduct, but did not suspend or cancel his license.

The Association, among other reasons, appealed on the ground that the license was not suspended or cancelled. There was a cross appeal by the respondent. On appeal the court held that the advertisements would be taken by the ordinary reader as indicating that the dentist had special qualifications. The principle is that "the opinion of his professional brethren of good repute and competency" is the guide for determining the propriety of a member's conduct. The Court, however, is entitled to consider the reasonableness of their opinion. In the present case three practising dentists stated they considered the advertisements unbecoming and unprofessional. The Court held that there was no ground for questioning the reasonableness of that opinion and maintained the appeal.

(G.V.V.N.)

1. Alberta Supreme Court, Appellate Division, reported in (1930) 3 D.L.R. 652.
2. 1927 (Alta.) c. 47, section 2.

Abstracts from Current Literature

Medicine

The Increase in Coronary Disease and Its Cause.
Denny, F. P., *New Eng. J. Med.*, 1936, **214**: 769.

Before 1930 there was no subdivision for coronary disease in the International Classification of Causes of Death. To make figures available for coronary disease previous to 1930 the writer reclassified according to the 1930 Classification all the deaths from heart disease in Brookline, Massachusetts, since 1900. Brookline had a population of 19,935 in 1900; this increased to 50,319 in 1935. The population shows some ageing, but this is not sufficient to explain the increase of over 100 per cent in the total death rate from all forms of heart disease during this period or the very great increase in coronary disease. In 1935 the rate of deaths due to coronary disease was 811 per 100,000 men over 45, and for women of the same ages, 333. Actually the death rate is even higher, as there are still many deaths due to coronary disease not diagnosed as such.

Osler and others have called attention to the relative freedom from coronary disease of those doing manual work. Those who suffer least from coronary disease have occupations which entail a certain amount of daily physical activity, while those who suffer most have sedentary occupations which require a mini-

mum of physical activity. Is not the sedentary life, the insufficient muscular activity, the basic underlying cause? It does not seem to the writer that the practice of medicine today entails any more strain than it did twenty-five years ago, and we cannot explain the increase in coronary disease in our profession in that way. The striking change in this period is the lessened physical activity of men. One often sees reference in the literature to the frequency of coronary disease among golfers. At first sight this seems inconsistent with the theory that physical inactivity favours the development of coronary disease. Golf is not *daily* exercise. Golf should be an ideal form of exercise for middle-aged men, if golfers took regularly some other form of exercise, preferably walking, when they were not playing golf. While we can do little to reduce worry and nervous strain, we can control our physical activities. Once it is recognized that coronary disease results in part, at least, from lack of muscular activity we have a definite point of attack. To prevent coronary disease a campaign of education is needed to teach the people that *daily* exercise is one of the essentials of health.

LILLIAN A. CHASE

A Note on the Common Occurrence of Serious Involvement of the Heart in Hyperpiesia. White, P. D., *New Eng. J. Med.*, 1936, **214**: 719.

Essential hypertension (hyperpiesia) is compatible with seemingly good health in the first few years of its existence; in that period it is often an accidental discovery. If persistent, it usually leads to heart failure. In fifteen years among 5,808 patients with symptoms or signs of cardiac abnormality there were 1,249 cases with hypertension (22 per cent); 51 per cent of these cases were males and 49 per cent were females; 68 per cent were between 50 and 70 years old. Angina pectoris was present in 329 of the cases (26 per cent) when they were first seen. Coronary thrombosis had occurred in 99 cases (8 per cent) and was a later complication in many more. Rheumatic heart disease was definite in 43 cases, with mitral stenosis in 23 of them. Cardiovascular lues was clearly present in only 10 cases and thyrotoxicosis in 6. Two patients had dissecting aortic aneurysms, as proved by autopsy. Diabetes was present in 18 cases and gout in 7. Seventy-four had had apoplexy and a number more developed this later.

The causes of death were determined in 100 consecutive cases; 95 died "cardiovascular-renal" deaths; 53 from congestive failure, 20 suddenly; 11 from acute coronary thrombosis; 6 from apoplexy; 5 from uræmia. Five died as a result of infection, including 3 from pneumonia.

The ages at death in this series of 100 cases were between 30 and 40 in 2, between 40 and 50 in 5, between 50 and 60 in 23, between 60 and 70 in 42, between 70 and 80 in 26 and over 80 in 2. Thus 72 per cent died before the age of 70.

To discover the onset of high blood pressure, to study it then, and to begin whatever sedative or other therapy seems effective, should be one of the aims of the practitioner. To accomplish this nothing short of annual examination of all adults is possible. There has been a tendency in the post-war period to swing too far from the old extreme of great fear of high blood pressure to the other extreme of reassurance and neglect. We should face the facts and take a middle course. LILLIAN A. CHASE

Achrestic Anæmia. Israels, M. C. G. and Wilkinson, J. F., *Quart. J. Med.*, 1936, **5**: 69.

The authors here describe and clearly differentiate from pernicious and certain allied anæmias a new type of primary megalocytic anæmia which they have named "achrestic anæmia" (from the Greek—"to utilize"). Pernicious anæmia is now generally regarded as being due to failure of maturation of the precursors of the red cells, due to the absence of a specific anti-anæmic factor, produced normally in the stomach by the interaction of a gastric ferment, hæmopoietin, with an extrinsic factor associated with the protein of the food. The achylia which is responsible for the absence of hæmopoietin in cases of pernicious anæmia is regarded as practically essential to the diagnosis. In other pernicious-like anæmias, the anti-anæmic factor is deficient for other reasons—in tropical nutritional anæmia, from deficient supply of extrinsic factor, in sprue, etc., from defective intestinal absorption. In achrestic anæmia, however, there is free hydrochloric acid and ferments in the stomach, and an adequate anti-anæmic principle has been demonstrated in the liver at post-mortem. Four cases of achrestic anæmia are fully described and a number of doubtful cases from the literature discussed. It is pointed out that such cases may frequently have been misdiagnosed aplastic anæmia. At post-mortem, the bone marrow is far from aplastic, the morbid anatomical changes resembling those of pernicious anæmia. The course is always prolonged, a characteristic feature being the failure, in varying degrees of initial completeness but eventually total, to respond to specific anti-anæmia therapy. Hence, there is finally an invariably fatal outcome, despite transfusions.

The authors believe that this rare type of anæmia is due to failure to utilize or mobilize from the tissue stores the anti-anæmic principle necessary for proper maturation of the red cells. They appear to have satisfactorily established "achrestic anæmia" as a definite, recog-

nizable clinical and pathological entity. It is to be hoped that they will be equally successful in discovering a method of treatment.

W. FORD CONNELL

Surgery

Osteomyelitis of Infants. Green, N. T. and Shannon, J. G., *Arch. Surg.*, 1936, **32**: 462.

The authors review a series of 95 cases of osteomyelitis in children under two years of age, at the Children's Hospital, Boston. In 82 of the 95 cases the lesion was acute and in 13 chronic osteomyelitis. The mortality rate was 21 per cent in the entire series. In those under six months of age the rate was 45 per cent. In infants over six months and under two years of age the mortality rate was 14 per cent. The disease is not rare in infants, which is contrary to accepted opinion. The femur was involved most frequently. In most instances the rapidly growing end of the bone was involved.

Fifty-two patients had antecedent extraosseous infections. The most common were infections of the respiratory tract, which occurred in 28 cases. Antecedent cutaneous lesions were present in 13 and omphalitis in 4 cases. Only 16 had a history of local injury of minor degree. The *S. hæmolyticus* was the causative organism in 48 cases out of 76 in which cultures were obtained, and the *Staph. aureus* in 20. The mortality rate in the former type of infection was 20 per cent and in the latter 32. In 8 of the 13 cases of chronic-osteomyelitis cultures were made and in 5 *Staph. aureus* was present.

The early symptoms in infants are not essentially different from those seen in older children, and are those of an acute illness, with local pain and sensitivity of the part involved. Protection and muscle spasm were recognized earliest of the local findings. Tenderness at the metaphysis is not a reliable sign at this age. Local œdema comes on more rapidly in infants and is much more diffuse than in older persons. Sepsis of the joint and infection of the soft tissues will have to be differentiated. Roentgen examination is of more diagnostic aid for infants than for older children or adults. The average period of hospitalization for the 95 cases was six weeks. Among the acute cases, there were 71 in which there was surgical intervention and 11 cases in which treatment was non-operative. In general direct compression of the marrow spaces, carried out through a window of moderate size in the bone, represents the usual conception of the ideal treatment of osteomyelitis. In many instances a considerable interval of time elapsed before operation, and these patients progressed more favourably than those operated on very early. In the post-operative care the wound is packed with petrolatum gauze and the part immobilized with a plaster cast which is bivalved.

G. E. LEARMONTH

The Disk-Shaped External Semilunar Cartilage. Fisher, A. G. T., *Brit. M. J.*, 1936, **1**: 688.

A disk-shaped external meniscus, sometimes associated with a definite lesion of the cartilage, is a rare cause of symptoms of internal knee-joint derangement.

Three symptom groups appear: (1) a clicking knee has been present, but with no history of trauma; (2) definite trauma superimposed on a clicking knee; and (3) definite trauma without previous symptoms. The most typical group is the first. The clicking may be a thud and associated with a sudden jolt forward for the external tuberosity of the tibia when the knee is flexed. The anterior part of the external cartilage becomes palpable. Occasional slight effusion and tenderness may be present. The lesion occurs predominantly in young females. Bilateral lesions may be present.

Acute symptoms developing in a clicking knee of long-standing indicate a need for surgical treatment. Cases in which all symptoms are acute in onset are treated by manipulation and palliative measures. Should this fail, or should the lesion recur, operation is indicated. Early operation prevents osteo-arthritic changes which may result from the lesion.

The disk-shaped external semilunar cartilage is due to the persistence of a layer of mesoderm in the developing knee-joint. Normally this liquefies in the centre and condenses at the periphery. The lesion should be borne in mind when a young patient complains of trouble towards the outer side of the knee-joint, and particularly if both knees are involved.

STUART GORDON

Gynæcology and Obstetrics

The Rôle of Anæsthesia in the Production of Asphyxia Neonatorum. Eastman, N. J., *Am. J. Obst. & Gyn.*, 1936, **31**: 563.

Chloroform when administered to mothers at the time of delivery has no demonstrable effect on the oxygen saturation of the fetal blood, but, because of its toxic effects on the mother, it is not to be recommended as an obstetric anæsthetic. Ether anæsthesia produces a slight depression of the oxygen saturation of the fetal blood, but this is not of sufficient degree to injure the fetus through anoxæmia. Nitrous oxide oxygen mixtures administered to mothers in proportions of 85:15 or weaker, and for periods of less than five minutes, regularly cause moderate degrees of fetal anoxæmia, but the normal full term infant is apparently not harmed. When nitrous oxide oxygen is given in concentrations of 90:10 or stronger over periods which exceed five minutes marked degrees of fetal anoxæmia are produced in about one baby out of three, and occasionally profound asphyxia neonatorum results.

ROSS MITCHELL

The Probable Rôle of the Hypercholesteræmia of Pregnancy in Producing Vascular Changes in the Placenta, Predisposing to Placental Infarction and Eclampsia. Bartholemew, R. A. and Kracke, R. R., *Am. J. Obst. & Gyn.*, 1936, **31**: 549.

The hypercholesteræmia of pregnancy is probably a normal physiological response to meet the requirements of rapid cell growth in the fetus and to prepare for lactation. An excessive degree of hypercholesteræmia is probably due to hyperpituitary or hyperthyroid activity, and is further increased by a diet rich in cholesterol-containing foods. Hypercholesteræmia is probably the fundamental basis for the toxæmias of pregnancy, and probably is responsible for the nausea and vomiting of pregnancy. Excessive storage in the liver in the first half of pregnancy is probably the cause of excessive fatty change in the periphery of the liver lobules with subsequent central necrosis in the inner zone of the lobules, which is so consistently found in fatal cases of pernicious vomiting of pregnancy.

Excessive storage in the placental arteries with resulting vascular changes is the predisposing cause of infarction in the placenta. Infarction arises from thrombosis or rupture of a placental artery. Acute or subacute infarction in the placenta results in autolysis of the affected tissue with the liberation of peptone, guanidine and histamine as toxic split-products of placental protein. The amount and location of the infarction, the degree of vessel obstruction, and the rapidity of autolysis determine whether pre-eclampsia, eclampsia or abruptio placentæ will occur.

If hypercholesteræmia is the fundamental basis for the toxæmias of pregnancy, the effect of excessive secretion of cholesterol into the intestinal tract through the bile and excessive storage of cholesterol in the endothelial system should be neutralized by the administration of thyroid extract or iodine, and by restricting the use of fats and cholesterol-containing foods during pregnancy.

ROSS MITCHELL

Exposure to the Violet Ray as a Prophylactic Agent to Nipples in the Last Month of Pregnancy. Maclellan, E. K., *J. Obst. & Gyn. Brit. Emp.*, 1936, **43**: 290.

One of the most common and most distressing complications of the puerperium is cracked nipples, and treatment has always been unsatisfactory. Exposure of the nipples to the violet ray, three times a week is advised, giving 6 or 8 treatments in all. Fifty cases are reported, in not one of which was the slightest difficulty in nursing encountered.

ELEANOR PERCIVAL

Ophthalmology

Inflammatory Exophthalmos in Catarrhal Disorders of the Accessory Sinuses. Cohen, W. *Arch. Ophthal.*, 1936, **15**: 457.

At times nasal sinus infection extends to the orbit, and when it does the condition is serious, not only for the eye but for the life of the patient. The inflammatory reaction in the orbit may be severe or mild. The severe acute and subacute infections of the orbit require conservative treatment, unless focalization and suppuration are evident. Chronic inflammatory infections of the orbit associated with exophthalmos are rare. In these cases the condition has a protracted course, and exploration of the orbital cavity does not often reveal any obvious cause for the marked exophthalmos that exists. In cases of long-standing exophthalmos it is interesting to observe that although the nasal sinuses are not a cause of clinical complaint pronounced and distinct pathological changes are regularly found. The surgical exploration of the orbital cavity has shown the presence of indurated tissue, and microscopic analysis of such tissue demonstrates a condition which could be caused only by some type of pyogenic agent. The Kronlein operation was performed with success in two cases of chronic inflammatory exophthalmos. This operation corresponds to the procedure of trephining the skull in cases of increased intracranial pressure or to scleral trephining in cases of intraocular pressure.

S. HANFORD MCKEE

Keratitis of Nasal Origin. Magitot, A., Tille, H. and Dubois-Poulsen, *Ann. d'Ocul.*, 1936, **172**: 1.

The nasal origin of certain affections of the anterior segment of the eye is well recognized and is not now emphasized any more than optic neuritis caused by an infective focus in the nasal sinuses. Numerous detailed cases are cited and conclusions are drawn regarding (1) phlyctenular kerato-conjunctivitis of children and of adults; (2) sub-epithelial kerato-conjunctivitis in adults, and (3) keratitis in the course of follicular conjunctivitis which may be traced to a nasal origin.

In the first group the nasal lesions seem to influence by infection, by their position, and by their rôle as a focus; in the second class, on the contrary, the nasal lesions act by irritation and consequent nervous disturbances. The third group appear to be made up of both, that is, action from a focus and from nervous influences.

In the presence of clinical keratitis it is most important to examine the nose and treat all nasal malformations. In children we find adenoids and hypertrophied tonsils, removal of which assists in the cure. In adults, shrinking

the mucous membrane with cocaine can easily be done by the ophthalmologist, and will often give an indication concerning the origin of the condition. Further treatment should be completed by the rhinologist. In conclusion the authors believe routine nasal examination should be made in all patients suffering from corneal affections. S. HANFORD MCKEE

Urology

The Lymphatics of the Lower Urinary and Genital Tracts. Mackenzie, D. W. and Wallace, A. B., *J. Urol.*, 1935, **34**: 516.

This paper is an experimental study with special reference to renal infections. A considerable amount of work has been carried out on the problem of the relationship of inflammatory lesions in the pelvic organs to coincident bacterial infections in the upper urinary tract, and this work has been fully reviewed. Opinion is divided as to whether or not there is a direct lymphatic connection, and the object of this paper is to study the path of this transportation with relation to the kidneys. At the outset it may be noted that lymphatic currents in general follow the flow of blood in the blood vessels, and as there are no continuous vascular channels from bladder to kidney it is not reasonable to expect such communication.

The authors' work was carried out on rabbits. A careful technique was developed, in which it was possible to remove all the lymphatics of the retroperitoneal area without the disturbing anatomical relations. A series of experiments was carried out on the rôle of these lymphatics in the absorption of dye from some of the pelvic organs and the relation of the upward drainage to the kidneys. Their results showed that no absorption of dye took place from a healthy bladder mucosa and very little even after trauma. Dye injected into the trigone, around the ureteral opening and around the bladder neck, passed to the glands at the bifurcation of the aorta. Dye injected into the wall of the lower third of the ureter passed to the lumber glands and main lymphatic chain, and the same was true when dye was injected around the renal pelvis. Even breaking the continuity of the main lymphatic pathway did not lead to opening up of subsidiary periureteral lymphatic routes. The conclusion is that dye injected in any of these locations can reach the kidney only by the lymphatic chains following the great vessels, thus to the thoracic duct, and thence *via* the blood stream.

N. E. BERRY

Atony of the Prostate. Marion, G., *J. d'Urol. Méd. et Chir.*, 1935, **39**: 401.

This condition occurs in young men, usually of a neurasthenic disposition, who are suffering from nervous exhaustion. They complain of pains, aches and distressing sensations in the region of the perineum and anus, the escape of prostatic fluid during defæcation, disturbances of urination, and varying degrees of impotence. Examination reveals a smooth, uniformly enlarged prostate. The expressed secretions are normal, and complete examination will usually reveal nothing.

These cases should not be treated as prostatitis, as often happens, a circumstance which aggravates the neurasthenia. The patients must first of all be reassured, and treatment should be avoided which attracts attention to the prostate. The distress may be relieved by any ordinary sedatives, and the general physical condition as regards rest, etc., must be improved.

N. E. BERRY

Neurology and Psychiatry

Non-Suppurative Encephalitis. Shaver, R. J., *J. Am. M. Ass.*, 1936, **106**: 699.

This clinical entity, first described in 1887, appeared but seldom in the literature until the present decade. Rather than suggesting the appearance of a new disorder, this is probably attributable to the greater alertness of the clinician. The etiology is obscure. Following widely varied disorders, such as the exanthemata, pneumonia, herpes, exogenous poisons, and, more recently, acute rheumatic fever (Winkelman) little evidence of a common factor can be made out. Authorities differ even on the nature of the process. Levaditi regards it as a degenerative lesion, probably due to some unknown virus, while to Globus it is a true inflammation.

Autopsy findings show engorgement of the leptomeninges, with some distension of the subarachnoid space and flattening of the underlying convolutions. Section shows a striking picture, with the numerous hæmorrhages mainly in the white matter, varying in size from extensive extravasations to minute punctate lesions. The perivascular spaces are packed with lymphocytes. Involvement of the nerve cells appears only in the hæmorrhagic areas, and there is no true demyelinization. Clinically, the picture is protean to a degree. Onset is usually abrupt with headache, convulsions or vomiting. Meningeal signs are common. There is no characteristic neurological picture, cranial nerve palsies and pyramidal signs being the most common. The cerebrospinal fluid is under moderately increased pressure, with a moderate pleocytosis (lymphocytic) and heightened globulin and sugar content. Smears and cultures are consistently negative. The blood picture presents a moderate polymorphonuclear leucocytosis. The writer concludes by presenting 5 cases, and is unable to suggest any treatment save the intramuscular injection of citrated blood. G. N. PATERSON-SMYTH

The Problem of Nervous and Mental Sequelæ in Carbon Monoxide Poisoning. Shillito, F. H., Drinker, C. H. and Shaughnessy, T. J., *J. Am. M. Ass.*, 1936, **106**: 669.

The existence of neuropsychiatric sequelæ from carbon monoxide poisoning has long been recognized. The underlying process is unquestionably due to the anoxæmia of brain tissue. The pathological picture is that of perivascular and perineuronal œdema, with varying amounts of nerve-cell destruction and occasional hæmorrhagic areas due to diapedesis of red blood cells. There seems to be some predilection for the cortex and the corpus striatum. So much for the fatal cases; there are but little data concerning those surviving the initial acute insult.

The present writers studied 21,000 cases seen in the New York metropolitan area over a decade. Over the same period but 39 admissions to the mental hospitals were made on the basis of CO poisoning, constituting 0.05 per cent. Serious mental and nervous sequelæ, then, are rare to a degree. Analysis of these cases suggests that sequelæ followed only extreme degrees of exposure. Unconsciousness was an invariable occurrence, and no cases appeared subsequent to so-called "chronic" exposure. In 33 per cent a clear interval of 7 to 20 days preceded the onset of symptoms. The mental picture was that of a typical confusional psychosis. Neurological sequelæ ranged from slight exaggeration of reflexes to well marked Parkinsonism. Sensory disturbances and peripheral motor neuritides also occur. All these signs improved, but there was generally some residuum. In the total group of 43 cases, 23 recovered completely, 9 suffered permanent mental or nervous sequelæ, and 11 died. G. N. PATERSON-SMYTH

Therapeutics

Types of Œdema and Their Treatment. Christian, H. A., *New Eng. J. Med.*, 1936, **214**: 418.

The types of œdema are schematically summarized and treatment outlined. Pleural fluid is much less effectively reabsorbed as a result of giving diuretics than fluid in other places in the body. It is wisest to remove pleural accumulations by mechanical measures promptly. The patient should rest in bed and be given digitalis, followed by morphine, gr. ¼, in the evening.

Ascites and subcutaneous œdema in patients with cardiac insufficiency respond well to the proper diuretics and seldom need mechanical removal. The patient with chronic non-valvular heart disease, with or without hypertension, oftenest with regular rhythm but at times with auricular fibrillation, is the type in whom marked œdema occurs oftenest. Such patients respond well to diuretics.

Digitalis should first be given with fluid restriction; after this the xanthine diuretics may be effective. Theocin (theophyllin) is most effective. It should be given in two doses (4½ to 7½ grains) with half a glassful of water at 7 and 10 a.m. An interval of 48 to 72 hours between the administration of diuretic drugs is preferable, although at times daily dosage is advantageous. This applies both to the xanthine and mercurial diuretics.

If xanthine diuretics fail to give good results, mercurial diuretics should be given. They have the disadvantage of requiring dosage by a parenteral route, preferably the intravenous route, because intramuscular dosage may cause irritation. Subcutaneous dosage is too irritatting to be used. Three mercurial diuretics are available, novasural (merbaphen), salyrgan (mersalyl), and mercuperin. Of these novasural is the most likely to cause toxic symptoms, while salyrgan and mercuperin, though containing more mercury, are less toxic. It is the author's impression that salyrgan and mercuperin cause a greater diuresis than does novasural.

The maximum activity of these mercurial diuretics takes place when the reaction of the blood, and probably of the tissue fluids, is shifted toward the acid side. Therefore the mercurial diuretic should be preceded by 48 to 72 hours of quite large doses of acid salts, such as ammonium chloride, 60 to 120 grains, three or four times a day.

Recently a mercurial diuretic has been prepared in the form of a suppository effective when given by rectum. This is the highly complex organic mercury compound which is present in mercuperin, there combined with a xanthine substance. This obviates the necessity for intravenous dosage, but still requires the preliminary days on ammonium chloride. The dose in suppository form is five times the dose for intravenous use. Experience with mercurial diuretics shows that they may be repeated every three or four days for months with no bad effects.

In the ascites caused by cirrhosis of the liver the xanthine diuretics have been ineffective. The mercurial diuretics quite often cause a considerable diuresis, but rarely control the ascites, and mechanical removal must be done. Sometimes a large dose of urea causes good diuresis in a patient with cirrhosis of the liver.

In œdema of renal origin, the xanthine diuretics have almost no effectiveness. The mercurial diuretics may produce a marked diuresis. In those forms of nephritis in which there is marked nitrogen retention mercurial diuretics should not be used. In cases not responsive to the mercurial diuretics large doses of urea, 60 to 90 grains a day, may be effective. Urea should not be used where there is nitrogen retention. If urea fails to give diuresis, intravenous injections of 15 per cent solution of gum acacia, in amounts from 400 to 500 c.c. may be given. This form of treatment is applicable to any form of œdema due to lowered osmotic pressure in the circulating blood.

LILLIAN A. CHASE

Pharmacologic and Therapeutic Properties of Crystalline Vitamin C. Wright, I. S. and Lilienfeld, A., *Arch. Int. Med.*, 1936, **57**: 241.

Vitamin C has been isolated in a crystalline form—cevitamic acid—from adrenal medulla, corpus luteum and a great many articles of food. Antiscorbutic properties are present in the pure acid and not in any attached impurities. There are various opinions about its therapeutic value in numerous diseases, notably different types of scurvy, but fairly definite evidence supports its effectiveness in conditions caused by capillary fragility. The authors describe a test to measure capillary fragility which they have found very useful in diagnosing and guiding treatment.

Apparently many adults suffer vitamin C deficiency, some because of poor diet, and others because they do not seem able to make use of the vitamin present in their food. This latter group improve on intravenous administration.

Of eight patients who had capillary fragility with many symptoms, such as soft bleeding gums, purpura, wasting, intestinal disturbances, swollen joints, weakness, hæmorrhages under the skin and from mucous surfaces, some did well on cevitamic acid orally, others required it intravenously. In all cases improvement took place in the clinical picture as well as in the capillary fragility as shown by tests. In cases diagnosed purpura hæmorrhagica cevitamic acid did not act so well. It was also unsatisfactory in cases of hæmophilia.

P. M. MACDONNELL

Pathology and Experimental Medicine

The Carotid Sinus Reflex in Patients with Hypertension. Gammon, G. D., *J. Clin. Investigation*, 1936, **15**: 153.

The observation that a state of elevated blood pressure develops in animals after denervation of the carotid sinus and aortic arch has suggested that certain forms of hypertension in man might similarly be due to a failure of the depressor mechanism. Several studies by other workers have supported this view. It has been found that, in hypertensive patients, direct stimulation of the carotid sinus by digital pressure over the wall of the sinus causes a marked fall in blood pressure; however, alteration of pressure within the carotid sinus by digital compression and release of the common carotid artery below the sinus produces only minimal and negligible changes in the blood pressure and heart rate. It has been concluded, therefore, that in hypertension there is a certain degree of insensitiveness of the carotid sinus endings to their physiological stimulus and that this may be responsible for the elevated blood pressure.

Thirty-one cases of hypertension were selected. With the individual prone, the carotid sinus was compressed against the transverse processes of the vertebræ for 30 to 60 seconds. The common carotid artery was obstructed as low in the neck as was possible, attempting to avoid traction on the sinus. The carotid artery was obstructed for periods up to 2 minutes. Control observations were made by pressure on an indifferent part of the neck. Simultaneous determinations of blood pressure and the pulse rate were made before, during, and after each procedure. A sufficient number of observations were made to determine the typical reaction of the individual.

In the study of these 31 patients with hypertension of either the essential type or that associated with arteriosclerosis the carotid sinus nerve endings were found, in accordance with other observations, to cause a depressor reflex which stimulated directly the response to changes in the intravascular pressure within the sinus, but which, contrary to the findings of others, was in the direction usually obtained in normal persons. These observations indicate that a failure of the carotid sinus mechanism was not the cause of hypertension in these patients.

JOHN NICHOLLS

A Study of the Lower Lobe of the Lung. Levitia, J. and Brunn, H., *Arch. Int. Med.*, 1936, **57**: 649.

In addition to the lungs being divided into lobes there is a physiological and anatomical division of the lower lobes into superior and inferior parts. This is corroborated by studying the embryology and perhaps more important the distribution of the bronchial tree. The superior division shows on an x-ray film as a triangular area posteriorly placed, with the apex towards the hilus. There is a separate division of the bronchial tree, a main stem which supplies this area, and this bronchus, because of its vertical position with the body in the prone position, is perhaps most apt to be the pathway for debris and various sources of sepsis. Pneumonia is said to involve this part more than any other, giving physical signs over the surface between the vertical border of the scapula and the spine, from the 4th, to the 8th, rib. The point is made that pneumonia of the superior division has been diagnosed encapsulated fluid and operation attempted. The rather inaccurate term "central pneumonia" has been also used rather loosely. When the inferior division is involved the x-ray shadow may be continuous with and masked by the liver shadow. Cases with roentgenograms are given to illustrate these points.

P. M. MACDONNELL

Some Experiments with Protamine Insulinate. Lawrence, R. D. and Archer, N., *Brit. M. J.*, 1936, **1**: 747.

The authors have carried out certain clinical experiments with Dr. Hagedorn's preparation of protamine insulin, and have been able both to confirm and to amplify previous published experiences. Whereas the duration of action of P.I. has been vaguely stated to be "twice as long" as ordinary insulin, the authors found that a massive dose (120 units) can act for more than two days (55 hours). Protamine insulinate is much weaker in dealing with carbohydrate food, and was not found to be capable of preventing hyperglycæmia after meals which contained any appreciable amount of carbohydrate. Its action on endogenous sugar, however, was nearly as good and as rapid as ordinary insulin. Since any degree of hypoglycæmia resulting from its use is induced much more slowly than with ordinary insulin, it causes much less symptoms of hypoglycæmia, even at the same blood sugar concentrations. When hypoglycæmic reactions were noted they occurred many hours after carbohydrate meals, usually between 3 and 6 a.m.

There is certainly very slow absorption of protamine insulinate from the subcutaneous tissues. It therefore has qualities which promise great things in severe diabetics with oscillating blood sugars and rapidly recurrent ketosis. On the other hand, it is certainly not strong enough in its action to control ingested carbohydrate, so that the authors subscribe to the conclusion previously reached by Hagedorn and others that it is advisable to give ordinary insulin before carbohydrate meals, and protamine insulin in the evenings, if full control of the diabetic condition is to be achieved. It is to be hoped that another preparation combining the qualities of both ordinary and protamine insulin may soon be developed.

W. FORD CONNELL

Hygiene and Public Health

An Appraisal of the Lead Hazards Associated with the Distribution and Use of Gasoline Containing Tetra-ethyl Lead. The Occupapational Lead Exposure of Filling Station Attendants and Garage Mechanics. Kehoe, R. A., Thamann, F. and Cholak, J., *Indust. Hyg. & Toxicol.*, 1936, **18**: 42.

The authors report on several studies undertaken to determine whether a hazard exists in the case of filling station attendants and garage mechanics exposed to tetra-ethyl lead gasoline.

1. Two young men who had had no experience in the handling of gasoline were studied for a period of four months with respect to their ingestion of lead in food and their excretion of lead in urine and fæces. Blood examinations for stippling and hæmoglobin were made. They were then put to work at a filling station dispensing tetra-ethyl lead gas, and similar observations made over a period of four months. No sign of increased lead absorption or excretion was found.

2. Similar observations were made on two young men before and after working as garage attendants in a garage using tetra-ethyl lead gasoline, and again no evidence of increased absorption or excretion of lead was found.

3. Two filling station attendants who had handled large quantities of leaded gasoline for a period of years when removed from employment and studied over a period of four months failed to show any change in lead excretion.

4. Two garage mechanics who had been occupied for years in garages using leaded gasoline showed no significant change in lead excretion.

The authors in previous papers have given evidence to show that most individuals ingest and excrete small quantities of lead. Evidence of occupational exposure to lead must therefore be found in significant increases in lead absorption or excretion.

FRANK G. PEDLEY

An Investigation of the Morbific Properties of Ethyl Gasoline. Lind, G., *J. Indust. Hyg. & Toxicol.*, 1936, **18**: 37.

This is a report of the results of an examination of 215 tank attendants, chauffeurs and mechanics exposed to ethyl gasoline in Denmark. The author states that ethyl gasoline was not introduced into Denmark until 1933 and that the sale has not been especially large. The 215 men were subjected to a careful clinical examination. Blood smears were taken and examined for stippled cells. No evidence of lead absorption or poisoning was found.

FRANK G. PEDLEY

The Mineralogical Composition of Air-Borne Foundry Dust. Hatch, T. and Moke, C. B., *J. Indust. Hyg. & Toxicol.*, 1936, **18**: 91.

The authors consider that samples of dust taken from rafters or other surfaces where dust settles are not typical of dust actually breathed by the workers. In this study, therefore, they have used samples collected by means of a paper filter bag and exhaust fan, and taken from the breathing level of the workmen. From the standpoint of the production of silicosis it has been demonstrated that the particles of small size (3 microns or less) are those chiefly effective, and that free silica ($S_1\ O_2$) is unquestionably the most dangerous constituent of the dust. This study indicates that in foundry practice sand chippers and sand blasters are those most exposed to dust of a dangerous type.

FRANK G. PEDLEY

Obituaries

Dr. Campbell Palmer Howard. Coming entirely unexpectedly to his wide range of friends here in Montreal, the death occurred on June 3, 1936, in California of Dr. Howard, one of the best known physicians of Montreal. He was delivering a series of lectures at the University of California and while there developed phlebitis, which ended with pulmonary embolism. He died at Santa Monica in his 60th year.

Son of an eminent Montreal physician, Robert Palmer Howard, Dr. Howard followed in his father's footsteps. He was educated at the old Montreal High School and graduated in both the faculties of arts and medicine at McGill University, obtaining his M.D. in 1901. He interned at the Montreal General Hospital and then went to Johns Hopkins Hospital, Baltimore, Md., where he served under Sir William Osler, McGill's most famous graduate.

After four years at Johns Hopkins, he did postgraduate work in the larger medical centres of Europe including Munich, Paris, Berlin and London and upon returning to Montreal was appointed physician to the outpatients department at the Montreal General Hospital. In 1910 he was made professor of medicine at the University of Iowa. He held this chair until 1924 and in the war served with No. 3 General Hospital (McGill).

He returned to Montreal in 1924 to become physician to the Montreal General Hospital and a professor of medicine at McGill University, posts once held by his father who also served as dean of the faculty of medicine at McGill from 1882 to 1889.

Many honours came to Dr. Howard during his years of practice and teaching. Among these was his election as president of the Association of American Physicians. He was a fellow of many medical societies and also became widely known through a steady output of articles written on his chosen subjects. He was, at his death, a representative of the faculty of medicine on the McGill Senate.

He is survived by his wife, formerly Miss Ottilie Frances Wright of Ottawa; a son, Palmer Howard, a medical student at McGill; a daughter, Miss Muriel Howard of Montreal, and a sister, Mrs. F. B. Futcher of Baltimore, Md.

Appreciations

Campbell Howard was a rock of a man. What he said, he said; and he meant it. What he was, he was; and there was no doubt about it.

When a nickname sticks to a man, it's more than a nickname, it's a stamp of his character, and when some student referred to Campbell Howard as "The Moose," he wrote his biography. Big, powerful, direct, with his head high, he went straight to his objective without subterfuge and without fear, and took no heed of cracking branches or splashing water.

Born in Montreal (in 1877), the youngest son of Dr. R. Palmer Howard, dean of the Medical Faculty of McGill University, teacher of Sir William Osler, and the most eminent physician of his day in Canada, Dr. Campbell Palmer Howard graduated in Arts and Medicine (1901) at McGill and after several years of postgraduate training, was called to be Professor of Medicine in the State University of Iowa, where he made a wide reputation as a physician and a teacher, and on the retirement of Dr. F. G. Finley as Professor of Medicine at McGill and physician of the Montreal General Hospital he returned to Montreal to succeed him.

He brought with him the spirit and the traditions of Osler, whose assistant he had been, and in addition brought a great store of energy and an immense capacity for work, and not only upheld but added to the reputation of the Montreal General Hospital as a centre for clinical teaching. He inspired his colleagues and his students with an ambition to do sound, thorough and honest work, and the effects of this inspiration have spread in a widening circle over the continent.

The worth of a medical man is best seen in the regard in which he is held by his colleagues, who are best fitted to judge him, and this regard was given to Dr. Howard in a large degree. He was invited far and near to speak at medical gatherings, and had been given the presidency of the Montreal Medico-Chirurgical Society, the Society for Clinical Investigation, and the Association of American Physicians.

As a student he was known as a football player, and in the later "nineties", the famous scrimmage trio of Ross, Howard and Grace, brought much fame to McGill.

But it is as a man and a friend that he will be remembered wherever rugged honesty and unswerving loyalty are held to be the marks of a man. To no man could Kipling's lines on Lord Roberts be more fitly applied—

> Clean, simple, kindly, well beloved,
> Flawless in faith and fame;
> Whom neither ease nor honours moved
> A hairsbreadth from his aim.

A. H. GORDON,
McGill University, Montreal.

Dr. Campbell Palmer Howard was called to the Chair of Internal Medicine of the State University of Iowa at a transitional period in the life of the College of Medicine. It was at a time when medical education in the United States was entering a phase of development based upon the practice of medicine as a science rather than as an art.

Dr. Howard brought to this mid-western University a new and stimulating spirit in medicine with a background of sound scientific training and outstanding personal and professional qualities. It was his great accomplishment to develop a solid and abiding tradition in the College of Medicine at Iowa as his great preceptor, Osler, had at Johns Hopkins.

The Howard tradition in Medicine at Iowa was based upon a painstaking thoroughness of study of the individual patient, a proper correlation of the laboratory findings with those obtained by methodical physical examination, and intelligent adaptation of clinical and laboratory research to the problems of disease, and, above all, the constant maintenance of the human relationship between the physician and his patient. The medical student who passed through his classes during his fourteen years at Iowa, the internes who were privileged to work under his direction in the University Hospital, the members of the medical faculty who served with him during these memorable and significantly formative years, will never forget nor cease to be influenced by his complete personal integrity, his unyielding professional idealism. He was an inspiring teacher, a wise and constructive leader, an outstanding clinician, a constant, sympathetic and understanding friend.

The Fates were kind when they decided that he should spend his last days in professional activity, engaged in the sort of work which he loved and for which he was so well fitted, and that he should pass away in the California home of his intimate friend, Dr. Charles J. Rowan, who, as professor of surgery, had been so closely associated with him for many years in building the Iowa tradition of medicine.

H. L. BEYE,
State University of Iowa, Ia.

The Chirurgical Society writes: "The Montreal Medico-Chirurgical Society has lost one of its most distinguished members. A past president, a member of its board of trustees and always a wise counsellor, he was devoted to its interests and was assiduous in his attendance and active participation in the meetings of the society, to whose younger members he was a constantly stimulating force. The society is deeply grieved over his passing at a comparatively early age, when his able and vigorous leadership still offered so much to his colleagues and the community in which he lived and worked."

Dr. G. G. Corbet, of Saint John, died on June 8 at his home following a three weeks' cardiac illness, the second of two such attacks from which he has suffered in the last few years. He leaves a wife and a son and daughter. The son is Dr. A. A. G. Corbet, also practising in Saint John.

Dr. Corbet was born in Saint John County May 12, 1870. He was a graduate in medicine from McGill (1898) and a Fellow of the American College of Surgeons. At the time of his death he was medical referee for the Workmen's Compensation Board (New Brunswick) and a consultant in Orthopædics for the Saint John Tuberculosis Hospital. He was a Rotarian and had done a large amount of work for crippled children under the auspices of this service club. He was also a member of the Canadian Club, the Clan McKenzie, St. Andrew's Society, Independent Order of Foresters, and a past-president of the Saint John Branch of the Canadian Legion, B.E.S.L. He was always a staunch liberal, and a Mason.

Dr. Corbet had a close connection for many years with the Canadian Army Medical Corps. He served overseas and at the end of hostilities was in command of No. 14 field ambulance, C.E.F. He had intended to retire from the position of District Medical Officer for Military District No. 7 on July 1st, and it was in this capacity that he has earned the highest regard of his military and medical colleagues from the fact that the Canadian Army Medical Corps Group in New Brunswick is one of the most completely, satisfactorily and harmoniously organized in the Dominion. This Army Medical organization comprising as it does a staff of seniors with much overseas experience, as well as an enthusiastic and growing group of young practitioners who have been willing to give ample time to prepare themselves in organization and practice, is a monument of which Dr. Corbet himself would have been indeed proud.

A. STANLEY KIRKLAND

Dr. Joseph McAndrew, of Georgetown, Ont., for 15 years a medical practitioner in Georgetown and a life-long resident of Halton County, died on May 15, 1936, in the Toronto General Hospital after a short illness. He was 65 years of age.

The late Dr. McAndrew was born in Norval and had taught school there for several years before entering university. Some years ago he left his practice in Georgetown to take up post-graduate studies in Germany, and later returned. He is survived by a sister and a brother.

Dr. Hugh Angus McColl, of Milton, Ont., who had come to Toronto to attend the Officers of Medical Health Convention in the Royal York Hotel, Toronto, died in his room on May 14, 1936, from heart trouble. He was 73 years old.

Dr. McColl was born in the Scotch Block in Halton County, and had been in practice in Milton for forty-seven years, serving in turn as Medical Health Officer and Coroner. He was at one time Mayor of the town. He was M.B. (University of Toronto, 1889).

Dr. Peter Duncan McLean, of Woodbridge, Ont., died on April 4, 1936, in his 80th year.

Dr. McLean was born in Oro Township, Simcoe County, his parents being pioneers of the county. He attended the local public school and Barrie Collegiate and graduated in medicine in 1879 (Toronto University) and commenced practice at Mono Centre at the age of 23 and in May of 1881 came to Woodbridge. He served on the Woodbridge Council and was Medical Officer of Health for that place and Toronto Gore for many years, and was the one time Liberal Member for the old Federal riding of Centre York. His wife, who before marriage was Miss Mary Denton, sister of the late Judge Denton, died in 1912.

Surviving are a son, Dr. G. D. McLean, Woodbridge; and a daughter, Ann, of Toronto.

Dr. Arthur L. Murphy, of Toronto, a well-known physician, and a member of a pioneer family, died at the Western Hospital on May 13, 1936, in his 73rd year. He had practised in Toronto for more than 35 years. Born at Rosemount, Ont., he was educated at Collingwood High School, Orangeville Collegiate and Trinity College Medical School, Toronto (M.D., C.M., 1892), taking post-graduate work in London, England. He was assistant superintendent at Sudbury Hospital before he commenced practice in Alliston. Later he moved to Toronto and built up an extensive practice in the western part of the city. He had been in failing health for the last two years. His wife, Elizabeth L. Cumberland Murphy, is his sole survivor.

Dr. Robert White Naylor, of Toronto, died suddenly on April 16, 1936, in his 47th year. He was a graduate of the University of Toronto (1915). The late Dr. Naylor had been a member of the medical staff of the Sun Life Assurance Company in Toronto. He is survived by his widow, a son and a daughter.

Dr. Wesley Herbert Secord died at his home in Winnipeg, on May 13, 1936, at the age of fifty-three. A son of Dr. Secord, of Brantford, and a brother of Dr. E. R. Secord, surgeon of that city, Dr. Secord was born and received his education at Brantford and McGill University, graduating in medicine in 1903. He practised as ship's doctor for two years and for a similar period at St. George, Ont. In 1907 he came to Winnipeg, where he soon acquired a good practice. In 1916 he went overseas with the 11th Field Ambulance, raised by the western universities, and saw nearly three years' service in France, where he was appointed Major and received the Military Cross for service at Paschendaele. After his return he served several years as Commanding Officer of the C.O.T.C. in Manitoba University.

Dr. Secord had a high sense of public duty and gave much to the medical profession. For eight years he was treasurer of the College of Physicians and Surgeons of Manitoba. In 1935 he was chosen to represent that College on the Medical Council of Canada. In 1921 he was President of the Winnipeg Medical Society; for several years he was on the Executive of the Manitoba Medical Association and he was Lecturer in Surgery in the Faculty of Medicine, University of Manitoba. He was also active on the Vestry and Advisory Board of St. Luke's Church and a member of the Masonic Order. He is survived by his widow, a sister of his closest friend. Dr. W. G. Campbell, and a son, Campbell, who is on the staff of the School of Economics, of London, England.

A friend writes of him as follows: "Herbert Secord was the soul of honour and of devotion to duty. He rendered great and meritorious service to the cause of organized medicine in Manitoba through his association with the College of Physicians and Surgeons, the Winnipeg Medical Society, and the Manitoba Medical Association, and on more than one occasion gave evidence of constructive medical statesmanship. He was an able surgeon, a warm friend, and a very gallant gentleman."

R.M.

Dr. William Ebenezer Waugh, of London, Ont., Bursar of the University of Western Ontario, died at his home on May 25, 1936. He was 85 years of age.

He was for many years a prominent medical practitioner and was a classmate of Sir William Osler at McGill University.

Dr. Waugh was born in London Township on May 23, 1851, a son of the late Christopher Waugh. He was educated at Moffat's Seminary in Komoka and McGill University, graduating in 1872.

He was one of the founders of the medical faculty of the University of Western Ontario and was lecturer and registrar for the faculty for 30 years. Also he was Secretary of the University Board of Governors. In

1919 he was granted the degree of Doctor of Laws, honoris causa, by his University.

Surviving are one daughter, Jean, at home, and one brother, Edward, of Komoka.

Dr. Robert Woods, of Leduc, Alta., passed away on April 24, 1936. He was a graduate of Manitoba University (M.D., C.M., 1905). He was one of the pioneer medical men of Alberta and practised for some years in the bush country twenty-five miles west of Leduc.

Dr. Sylvester Nash Young, of Ridgetown, Ont., died suddenly on May 19, 1936. He was born in Ridgetown in 1854, a son of the late Mr. and Mrs. Russel Young, who were among the earliest settlers of the district, coming to Ridgetown in 1830 from Vermont. He married Frances Nash. The doctor was the youngest of the family and the last survivor. He took his collegiate training at St. Thomas High School and taught school for a number of years both in Kent and Elgin. He took his medical degree in 1889 (Victoria). After graduating, he practised in Fletcher for 18 years, returning to Ridgetown 28 years ago. He had been Medical Officer of Health for Ridgetown during the greater part of his practice here. Dr. Young was a champion of child welfare work and in his capacity as Medical Officer of Health did a great part in the promotion of this work. Within the past month he assisted in obtaining for Ridgetown a tuberculosis clinic. Surviving are his widow, one son, William, of Whonnock, B.C.; and one daughter, Mrs. Lee Simpson of Ridgetown.

News Items

Great Britain

Royal College of Physicians of London.—At a meeting of this College held on April 30th Sir Frederick Banting and Dr. Harold Campbell Parsons, both of Toronto, were elected Fellows.

Gifts to Hospitals.—Lord Beaverbrook has given £24,900 to St. Mary's Hospital, Paddington. His gift of £63,000 in 1929 made it possible for the medical school to be rebuilt.

Sir Herbert Austin, Chairman of the Austin Motor Company, announced at the annual meeting of the Birmingham United Hospital recently that he would defray the cost, totalling £7,000, of a radium bomb to complete the equipment installed for the treatment of cancer.

Gift to the Royal College of Surgeons. — The Bernhard Baron Trust has made a gift to the Royal College of Surgeons of £25,000 for the provision of new research laboratories. At a meeting of the Council of the College recently a letter from the trustees was read offering the gift and asking that the new laboratories be named the Bernhard Baron Laboratories, and that publications of research carried out therein should bear an indication that the work had been carried out in the Bernhard Laboratories of the College. The Council passed a resolution accepting the gift and the conditions attached to it. The decision of the Bernhard Baron Trustees to make this gift testifies to their appreciation of the fact that by helping medical research they may be doing more for the relief of suffering from disease than by any of their direct gifts for charitable purposes.

Alberta

The annual Refresher Course at the University of Alberta, was held May 11 to 15, 1936. While the attendance was not as large as in former years the lectures were of a high order, and those present were greatly pleased. The smaller attendance was in a measure attributed to the bad roads, and the further fact that the Canadian Medical Association Convention will be held in Victoria, B.C., in June, when Alberta is looking for a good representation.

Physicians residing north, east and west of Edmonton, met in convention May 12th, and organized a district association of the Alberta Division of the Canadian Medical Association. This district is large and sparsely settled, from Lamont on the east to Jasper on the west, north to Athabasca, and north-east to Elk Point and Cold Lake. The following district officers were elected. Dr. T. W. E. Henry, Fort Saskatchewan, District Representative; Dr. R. A. Walton, Stony Plain, Vice-Representative; Dr. M. E. Tiffin, Edson, Member of Nominating Committee; Dr. J. H. Riopel, Legal, Secretary.

The Executive Committee of the College of Physicians and Surgeons met in Calgary May 7,1936, the following were present: Drs. A. E. Archer, Lamont; R. Parsons, Red Deer; R. B. Francis, Calgary; W. S. Galbraith, Lethbridge. The time was mostly occupied in devising ways and means for closer cooperation with the Provincial Medical Association.

Fire recently destroyed the Berwyn Hospital. This institution was under the Women's Institute, and its destruction will be a great loss to the community. Eight patients were in the hospital at the time but were removed to safety without injury.

A special committee was appointed by the four hospitals staffs in Edmonton, to discuss some satisfactory arrangement whereby patients coming in from the country who are unable to pay for their medical care and treatment may be assisted in part by the municipality from which they come. This matter has been under advisement with the College of Physicians and Surgeons and the Union of Rural Municipalities for some time, but as the matter rests with the individual municipalities themselves it is hard to get definite action during a depression period, when so many individuals are willing to pass on their own responsibilities to others. The medical men went on record that they were willing to go more than half way to meet the emergency.

The Board of Directors of the Canadian Medical Association, Alberta Division, are meeting in Calgary, June 3rd, to arrange for the date and program for the annual convention, which is to be in Calgary some time in September, Dr. D. S. Macnab is *President* and Dr. W. Fulton Gillespie is *President-elect*, and they are both active in their duties. G. E. LEARMONTH

British Columbia

The Health Insurance Act which was passed at the last session of the Provincial Legislature was proclaimed on May 18th, and steps are now being taken to set up machinery for its administration. Dr. Alan Peebles has been appointed chairman of the Health Insurance Commission, with W. T. Barrett, M.D., of Victoria to assist him. The Provincial Secretary has announced that offices for the Commission will be located in Victoria, and it is expected that Health Insurance will be put into active operation in approximately six months from the time of its proclamation.

Dr. G. F. Amyot, formerly Medical Officer of Health for the district of North Vancouver, has been appointed as assistant to Dr. H. E. Young, Provincial Health Officer, and will exercise a general supervisory

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position over the hospitals in the province, advising Dr. Young on hospital affairs.

A luncheon meeting of the British Columbia Medical Association was held at the Hotel Georgia in Vancouver on May 20th. It was attended by several members of the Association Executive from other parts of the province, who expressed on behalf of the physicians in their districts the most intense interest on the subject of Health Insurance and gave assurance of the solidarity of the profession in supporting the work being done on their behalf by the Health Insurance Committee of the College of Physicians and Surgeons. The meeting was addressed by Dr. Wallace Wilson, chairman of the Health Insurance Committee, *vice* Dr. W. E. Ainley who has been obliged to relinquish that position for reasons of health, and by Dr. M. W. Thomas, the recently appointed Executive Secretary of the British Columbia Medical Association. Dr. Wilson outlined the recent activities and attitude of the Health Insurance Committee in view of the present state of Health Insurance, and Dr. Thomas dwelt upon the very satisfactory results which were promised by the unity which now exists to a degree never before attained among the members of the profession in the province.

Dr. M. W. Thomas has left for a tour of the province preliminary to the annual meeting of the British Columbia Medical Association which will be held in conjunction with the meeting in Victoria of the Canadian Medical Association in June. It is expected that a large proportion of the medical profession in British Columbia will be present at this meeting. D. E. H. CLEVELAND

New Brunswick

Dr. L. M. Curren, Saint John, is again well enough to leave the hospital following several months hospitalization, during the course of which he underwent two abdominal operations. He expects to resume his practice shortly.

Mount Allison University, Sackville, honoured Hon. W. F. Roberts, conferring on him the degree of Doctor of Laws at their recent convocation. At the time of the presentation it was stressed that Dr. Roberts was the first Minister of Health appointed in the British Empire and was the founder of the present extensive Public Health Department of New Brunswick.

Dr. George Skinner was made a member of the American Association of Thoracic Surgeons at their recent meeting in Rochester. He attended at the same time the meeting of the American Surgical Association in Chicago.

Two of the local physicians in Saint John, who have been on extended holidays following cardiac disasters, are returning shortly to practise. Dr. W. E. Rowley and Dr. D. P. Mahoney are the fortunate gentlemen who have successfully combated serious illnesses during the past year.

Dr. H. A. Farris was elected President of the Saint John Canadian Club. A. S. KIRKLAND

Nova Scotia

The Annual Meeting of the Nova Scotia Medical Society will be held in Halifax, August 31st to September 4th in conjunction with the Dalhousie Refresher Course.

After twenty minutes' deliberation a Lunenburg, N.S., jury returned verdicts of "not guilty" on four charges of manslaughter brought against Dr. William P. Young and his wife, Lila P. Young, proprietors of the "Life and Health Sanitarium". Dr. Young is a chiropractor, his wife a licensed midwife. The sanitarium is an institution primarily for the care of unmarried mothers, located at East Chester. The case originated in February when the accused were arrested following the death of a male infant and its mother who were under their charge.

The prosecution attempted to prove, by the questioning of expert witnesses, faulty aseptic technique and the improper use of instruments in delivery. An expert witness for the defence supported the contention of the accused that the baby was stillborn, and praised the arrangements of the home. Mrs. Young, in her own defence, outlined the history of the institution and stated that 148 maternity cases have been delivered in the past eight years with an infant mortality of twelve and one maternal death. Most of these cases, it was pointed out, had no prenatal care before coming to the home.

Fourteen thousand dollars has been allotted for a new wing to the Middleton Hospital. This little institution has been serving a large rural community as well as the town, with a capacity hitherto limited to fifteen beds. When the Kentville hospital is erected the Annapolis Valley will be well equipped with a chain of small hospitals from Windsor to Digby, with a total capacity of about 150 beds.

Mrs. Sadie Hyland, of Halifax, is no richer for her $10,000 suit for damages against Proctor and Gamble Company. Mrs. Hyland claimed she developed a pharyngitis and laryngitis from the inhalation of fumes of Oxydol, a laundry soap. Expert medical testimony was called by both sides. Neither evidence carried as much weight with the jury as the soap company official who inhaled the steaming fumes of an Oxydol solution without suffering or injury.

Until the medical profession agrees on the causes of anthracosis and chalicosis they cannot be classed as industrial and compensational diseases, L. D. Currie, M.L.A., told a meeting of United Mine Workers at Glace Bay, N.S.

The medical profession and press are combining in an attempt to reduce the tuberculosis death rate in Cape Breton. The Cape Breton death rate is 4 per 100,000 less than the province as a whole. The provincial death rate was 91.0 per 100,000 in 1934 as against 104.6 in 1930. ARTHUR L. MURPHY

Ontario

Dr. William Edward Gallie has been appointed Dean of the Faculty of Medicine, University of Toronto, for a period of three years. Dr. Gallie, a surgeon of international reputation, and until 1929 surgeon-in-chief of the Hospital for Sick Children, latterly chief surgeon at the Toronto General Hospital and Professor of Surgery in the Faculty of Medicine, was born in Barrie in 1882. Educated in Barrie Collegiate Institute, he graduated to the University of Toronto and received his degree in 1903. He was appointed assistant surgeon in the Hospital for Sick Children in 1906. In 1920, and for nine years, he was chief surgeon. He served with the C.A.M.C. overseas from 1916-18, and is a member of the Rosedale Golf Club, his sole recreation. He is a Fellow of the following medical societies: Royal College of Surgeons of England, American College of Surgeons, Royal College of Surgeons of Canada, President of the American Orthopædic Association, American Surgical Society, International Orthopædic Society, International Society of Surgery.

The Charles Mickle Fellowship was awarded this year to Donald D. VanSlyke, Ph.D., Hon.Sc.D., of the Rockefeller Institute, for his methods for blood analyses and gasometric micro-analyses, his work on respiratory and renal functions, on diabetes and nephritis, and, in general, for his research on quantitative clinical chemistry. This Fellowship is awarded

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annually to that member of the medical profession who is considered by the Council of the Faculty of Medicine of the University of Toronto to have done most during the preceding ten years to advance sound knowledge of a practical kind in medical art or science.

Construction has already commenced on a $450,000 addition to St. Michael's Hospital, Toronto. This involves the demolition of part of the old building now used for administration offices. The new building will be eight stories high, and will probably be ready for occupation by January 1, 1937. Fifty beds will be provided, as well as new office quarters.

During the week of May 19th the patients and staff of Grace Hospital, Toronto, were transferred to the new buildings of the Toronto Western Hospital on Bathurst Street, thus completing the merging of these two hospitals and forty-three years of service of the present Grace Hospital on College Street.

The Canadian Red Cross Society has approved of the opening of two new hospitals in Ontario,—the first to be at Jellicoe, the jumping-off place for the Sturgeon River Gold Fields; the second in Haliburton, in a district which will serve approximately 2,900.

On Tuesday, May 12th, the birthday of Florence Nightingale, the Honourable J. A. Faulkner, M.D., Minister of Health for the Province of Ontario, in the presence of a large number of visitors, opened the new wing of the Cobourg Hospital.

During the week of June 22nd, the Kingston General Hospital celebrates the fiftieth anniversary of the founding of its nurses' training school.

The Hotel-Dieu Hospital at Cornwall has erected a new wing at a cost of $75,000. This contains three operating rooms, modern in every respect. With the opening of this new wing the Sisters of St. Joseph are celebrating the Tercentenary of the founding of their Order.

Dr. K. G. McKenzie, of Toronto, was elected, on May 17th President of the Harvey Cushing Society at its fifth annual meeting in Rochester, Minn.

Dr. S. J. Streight, of Toronto, presided at the twenty-sixth annual meeting of the Medical Section of the American Life Convention at White Sulphur Springs, West Virginia, June 11th, 12th, and 13th.

The fifty-sixth annual meeting of the Ontario Medical Association, held in London, May 26th, 27th, 28th, and 29th, was one of the most successful in its history. The medical registration was 1,122 in addition to 295 ladies. At the sessions of Council 117 doctors were present. The weather was ideal, papers and discussions were excellent, and the exhibitions, both scientific and commercial, were attractive and educational. The profession of Ontario owe a debt of gratitude to the London Committee for the excellency of the arrangements and the provision made for the comfort and entertainment of those present.

His many friends in Ontario will learn with pleasure that, on May 20th the University of Manchester conferred the degree of Doctor of Science upon Prof. J. B. Leathes, formerly of the University of Toronto.

At the recent meeting of the Ontario Medical Association at London awards were made to a number of exhibitors whose work was of special note and importance.

The gold medal for excellence of presentation of an exhibit was given to Dr. W. S. Stanbury, of Hamilton. A certificate of merit for the outstanding educational exhibit was won by the War Memorial Children's Hospital, London.

Other awards were:

For originality of investigation—silver medal, Dr. J. H. Hannah, Toronto; certificates of merit, Dr. G. S. Williamson, Ottawa; Dr. A. W. Ham, Toronto, and Dr. E. R. Secord and J. H. Moss of Brantford.

For excellence of presentation—silver medal, Dr. V. R. Lapp, Hamilton; certificates of merit, Dr. G. E. Richards and Dr. A. D. Irvine, Toronto; Dr. James Miller, Kingston, and Dr. T. G. M. Drake, Toronto.

J. H. ELLIOTT

Quebec

Brother Marie-Victorin, who is Professor of Botany in the University of Montreal, has made an interesting discovery, which he reported at the last meeting of the Royal Society of Canada. A MS dealing with some two hundred Canadian plants was discovered about seventeen years ago in the library of the St. Hyacinth Seminary by Msgr. E. Choquette. It was unsigned and its authorship had to be determined by internal and other evidence. Brother Marie-Victorin undertook this work, travelling even to France and Germany for the purpose. As the classification of the plants is pre-Linnæan the latest date for the manuscript could fairly accurately be determined. It has finally been proved that it is the combined work of two botanists of about 1707 to 1744—Michel Sarrazin, the king's physician at Quebec (who discovered the pitcher plant, and after whom it is named *Saracenia purpurea*) and his friend Sebastien Vaillant, of Paris, the successor of the great botanist Tournefort at the Jardin des Plantes. Sarrazin sent his notes to Vaillant and received in return certain information. The two sets of notes had evidently been put together by one of the early Jesuit priests teaching botany in Quebec for his own information. This man did not bother to rephrase the notes as he copied them, so that sometimes they read as if they had been written in France and sometimes as if written in Canada. The key to the authorship was eventually found to be in the MS itself. The numbers of the specimens of plants as recorded in the notes sent from Canada to France correspond with those of the same specimens referred to in Vaillant's notes where they are credited to Sarrazin.

For her exhibit on congenital cardiac disease, which displayed the greatest originality in scientific investigation, Dr. Maude E. Abbott of McGill University, was awarded a gold medal by the Ontario Medical Association on May 28th. Prizes to doctors for the best scientific exhibits at the annual medical convention were distributed at a dinner, attended by Premier Hepburn.

Dr. Abbott's exhibit consisted of photographs, drawings, paintings, charts, diagrams, and tracings having to do with congenital cardiac disease. It also included anatomical and pathological specimens showing various types of fish and reptilian hearts.

Dr. Abbott, who is assistant professor of medicine and curator of the museum of the history of medicine, is being superannuated this year under McGill's retiring plan. Dr. Abbott graduated in medicine from Bishop's College, then in Montreal, as McGill at the time did not permit women to take the medical course. Her training as an interne was done at the Montreal General Hospital and later McGill conferred on her an honorary medical degree. Dr. Abbott is a licentiate of the Royal College of Physicians and Surgeons (Edin.) and a Fellow of the Royal College of Physicians of Canada.

We are informed that the Chiropractor's Bill which was recently before the Quebec Legislature has been turned down. It met with much opposition from the various medical bodies in the Province, both French and English.

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General

The Second International Medical Week in Switzerland.—This course, organized by *Le Journal Suisse de Médicine*, under the distinguished patronage of the Swiss Federal Council, will be held from August 31st to September 5th at Luzern. The program will be sent to those interested on application to the Secretariat de la Semaine Médicale Internationale, Klosterberg 27, Bâle, Switzerland.

Among those taking part are the following. R. Bing, Bâle; P. Claimont, Zurich; P. Decker, Lausanne; G. Frontali, Padua; J. Fulton, Newhaven, U.S.A.; E. Gäumann, Zurich; R. Grégoire, Paris; Freih. E. v. Gröer, Lwow (Poland); H. Guggisberg, Bern; H. C. Jacobaeus, Stockholm; W. Löffler, Zurich; E. Mellanby, London; O. Naegeli, Zurich; M. Péhu, Lyons; E. P. Pick, Vienna; F. Rathery, Paris; M. Roch, Geneva; F. Sauerbruch, Berlin; Ph. Schwartz, Istanbul; A. Szent-György, Szeged, (Hungary); A. Vogt, Zurich; L. K. Wolff, Utrecht; H. Zangger, Zurich.

Membership is 10 Swiss Francs (about $2.00) and this includes subscription to the Transactions.

The International Congress on Hepatic Insufficiency. will take place at Vichy from September 16 to 18, 1937, under the Chairmanship of Professor Maurice Loeper, a Member of the Academy of Medicine, we give you below the names of the members of the Committee:

Honorary Presidents: Professors Von Bergmann, Berlin; Carnot, Paris; Mariano Castex, Buenos Aires; Dustin, Brussels; Walter Langdon Brown, London; G. Maranon, Madrid; Marchoux, Paris; Pende, Rome; G. H. Whipple, Rochester, N.Y. *President:* Prof. Maurice Loeper, of Paris. *Vice-Presidents:* Professors Cade, Lyons; Giraud, Montpellier; Berklen, Strasbourg; Olmer, Marseilles. *General Secretary:* Dr. J. Aimard, 24, Boulevard des Capucines, Paris (IXe), France.

The proceedings of the Congress will be divided into two sections: *Medicine and Biology:* Presided over by Professor Noël Fiessinger, Professor of Experimental and Comparative Pathology at the Faculty of Paris. *Therapy:* Presided over by Professor Mauriac, Dean of the Faculty of Bordeaux.

We would remind you that the Congress on Hepatic Insufficiency will be preceded by the *International Congress on Gastro-Enterology*, which will be held in Paris on September 13, 14 and 15, 1937.

Olympiad at Berlin.—Dr. Malcolm M. Crawford, Chief Coroner of Toronto, has been appointed official physician of the Canadian team attending the XIth Olympiad in Berlin from August 1st to 16th. Dr. Crawford will also attend the International Congress of Medical Advisers for Athletics to be held in Berlin from July 27th to 31st as official Canadian Representative.

The next meeting of the **Western New York and Ontario Urological Society** (branch of the American Urological Association) is to be held at Guthrie Clinic, Sayre, Pa., on October 10, 1936.

The American Public Health Association will convene in New Orleans, La., October 20th to 23rd, for its 65th Annual Meeting. The national headquarters are at 50 West 50th Street, New York, N.Y., and Dr. Reginald M. Atwater is Executive Secretary.

Book Reviews

Chronic Rheumatism. R. F. Fox, M.D., F.R.C.P., F.R. Met. S., President of International League Against Rheumatism, and J. Van Breemen, Honorary Secretary of International League Against Rheumatism. 364 pages. Price 12s. 6d. J. & A. Churchill, London, 1934.

By virtue of their extensive personal experience in rheumatic diseases and their work as officers in the International League Against Rheumatism the authors are able to present much valuable information on this vital subject in a small volume. They discuss four causal factors of rheumatism and certain physiological features, and give a concise clinical picture of several types of rheumatic disease. In addition, there is an outline of nomenclature, with various classifications and certain forms of treatment.

The four causal factors, briefly submitted are: (1) infection, local and general; (2) constitutional anomalies; (3) abnormalities and circulation of the skin and periphery, and (4) external factors, such as environment and occupation. The physiology of the skin as an organ of the body and changes in the peripheral circulation are stressed as factors of prime importance in the rheumatic diseases. In attempting to present a brief clinical picture of the main types of rheumatic disease, the authors have the usual difficulties in portraying clearly the various types as distinct clinical entities, with respect to the terminology, classification, differential diagnosis, treatment and prognosis. The problem of classification is demonstrated by a presentation of the various types of nomenclature now used in different countries, and a solution is offered toward unification of terminology through the agency and efforts of the International League. The treatment herein outlined is based almost entirely upon physio-therapeutic methods, and is more applicable to European countries where spas and mineral baths are more available to patients. The authors make no attempt to discuss the other important general or local measures of therapy. Also it must be stated that some types of arthritis are omitted entirely.

The tremendous social, industrial and economic features of rheumatic diseases are presented, showing the necessity and importance of coordinated international studies of the whole vital problem. In this respect the function of the International League Against Rheumatism, with its affiliated societies in the leading countries of the world, is shown to be of extreme importance. A brief review is added of the work being done in the various European countries, and suggestions are given for local organizations. This volume is submitted by the authors as a plea for further world-wide study of the problem of rheumatic and arthritic diseases, and, as such, should be read by everyone interested in this important branch of medicine.

An Introduction to Surgery. R. Morison, M.B., F.R.C.S.(Edin.), F.R.C.S.(Eng.), M.A., D.C.L., LL.D., Emeritus Professor of Surgery, Durham University, and C. F. M. Saint, C.B.E., M.D., M.S., F.R.C.S., F.R.A.C.S., Professor of Surgery, Cape Town University. Third edition, 367 pages, illustrated. Price $4.50. John Wright & Sons, Bristol; Macmillan, Toronto, 1935.

In the third edition of his Introduction to Surgery Professor Morison has again the assistance of his former pupil, Professor Saint, of Capetown. The book really makes an attempt to aid the student to think for himself and to correlate clinical findings and pathological changes. It is surgery studied from a thorough knowledge of the pathology of the conditions. It attempts to group diseases and to study them from certain definite principles. This is well exemplified in the study of inflammation and its results, which are the same, no matter what part of the individual is affected.

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Chapter one, on the basis of the principles of surgery, is an excellent introduction to the following chapters. It would be difficult to single out any one portion, but the chapters on causation on effects of inflammation, of gangrene, and of mechanical obstruction are particularly good, and the matter is presented in a clear, concise manner. There is a good chapter on the similarity between syphilis, tuberculosis and malignant disease. The book may be recommended without fear to every student or teacher.

Medical Papers Dedicated to Henry Asbury Christian. From his present and past associates and house officers at Peter Bent Brigham Hospital, Boston. 1000 pages, illustrated. Price $10.00. Waverly Press, Inc., Baltimore, 1936.

This volume of papers has been prepared as a token of regard presented to Dr. Henry Christian on the occasion of his sixtieth birthday by his present and past associates and House Officers at the Peter Bent Brigham Hospital. Collections of papers such as these have a historical value in the picture they give us of the men they memorialize. The outstanding feature of Dr. Christian's work is (for he is still a young man of 60) his training of men in clinical medicine. This presupposes unusual qualities of both scientific powers and a capacity for bringing out the best in others. The two are not necessarily always associated, and only rarely do they exist in such a degree as in Dr. Christian.

One looks in vain for the explanation of his power for thus influencing men. We are told of his own prodigious output of work on a variety of subjects; his powers of organization; his ceaseless driving energy. All these have been present in other men who cannot claim anything like the width of his influence. The quality which completes his personality must remain as indefinable as it is powerful. This book is a fine illustration of what the combination of these various qualities can do. It includes contributions on a very wide variety of medical subjects by most of those who have worked with him, and the quality of their writing is in general of a high order. The stimulating mental atmosphere of Dr. Christian's work is well brought out in the *Appreciation* by "W.T.V.", in which he speaks of the wealth of fresh ideas and suggestions which can be traced directly to the famous Saturday rounds at the Peter Bent Brigham. Where keen minds are drawn together by mutual attraction one expects to find this to happen. "Truth", as the theologian put it, "is the spark struck out in the clash of opposing opinions". But one must have such a man as Dr. Christian to bring such minds together.

Surgery: Queen of the Arts and Other Papers and Addresses. W. D. Haggard, M.D., F.A.C.S., D.C.L., Professor of Clinical Surgery, Vanderbilt University School of Medicine. 389 pages, illustrated. Price $6.00. W. B. Saunders, London and Phila.; McAinsh, Toronto, 1935.

Dr. Haggard from his long and varied experience has given us thirty-two addresses of historical or clinical or laudatory substance, and has combined the humanitarian with the clinical interests of his life. The art of surgery is the most imperative and the most important of the arts and is essentially constructive and re-creative. From the dawn of history it has increased in its scope of service and in the beauty of its performance. "The first requirement of the surgeon is a conscience". In Roman times a surgeon must have "a firm and steady hand, a keen eye and the most unflinching courage"; today "he must be a man of the broadest and tenderest sympathies and one whose kindliness and gentleness are unfailing". "Petrarch said that a surgeon unlettered in Anatomy carves the human body as a man carves wood".

Dr. Haggard gives us with startling clarity the clinical pictures of surgical conditions of the upper abdomen and outlines the diagnostic assistance of the x-ray. He counsels delay in delayed cases of acute appendicitis with complete laboratory work and adequate preparatory treatment. His experience with x-rays as preparatory and post-operative treatments for cancer of the breast have failed to convince him of their efficacy. He is fully conscious of the limitations of the surgeon in treating the neurotic, and regards surgery as "a very dangerous type of suggestion therapeutics".

This little book expresses Dr. Haggard's wide interests in his fellow man, medical and lay, and is well worth reading.

Obstetrical Practice. Alfred C. Beck, M.D., Professor of Obstetrics and Gynæcology, Long Island College of Medicine. 702 pages, illustrated. Price $7.00. Williams & Wilkins, Baltimore, 1935.

This textbook presents ample evidence of the sound, conservative practice for which Long Island College Hospital has been noted these many years.

It is difficult to write a treatise on obstetrics differing much from other recent publications but Dr. Beck has achieved this task. The outstanding characteristic of the book is his most excellent description of the mechanism of labour in all presentations. He, himself, has considerable facility in diagrammatic drawing, and in addition to this has had the assistance of a professional illustrator. As a result the book is illustrated in a most profuse manner. There are, for instance, 63 drawings illustrating the mechanism of labour in the L.O.P. position, and these are followed by a pictorial summary made up of 56 additional illustrations. The same profuseness of illustration is carried out in the case of the other positions. The book, therefore, will appeal particularly to the undergraduate student.

The text is necessarily somewhat abbreviated because of the many drawings, but points of particular importance are emphasized in bold-faced type, *e.g.*, "infected teeth are a menace, and pregnancy is not a contra-indication to their removal". On minor points some slight difference of opinion might exist. For instance, the author states that rectal examination during labour "because it may be done with very little risk to the patient" should be made by the beginner after every vaginal examination. It is questionable whether rectal examination is as free from risk as the author implies, and a double examination of each patient by the beginner might tend to make him feel that it could be done with impunity. Some obstetricians may also question the advisability of firmly packing the open cavity of a breast abscess with gauze which is left undisturbed for a week.

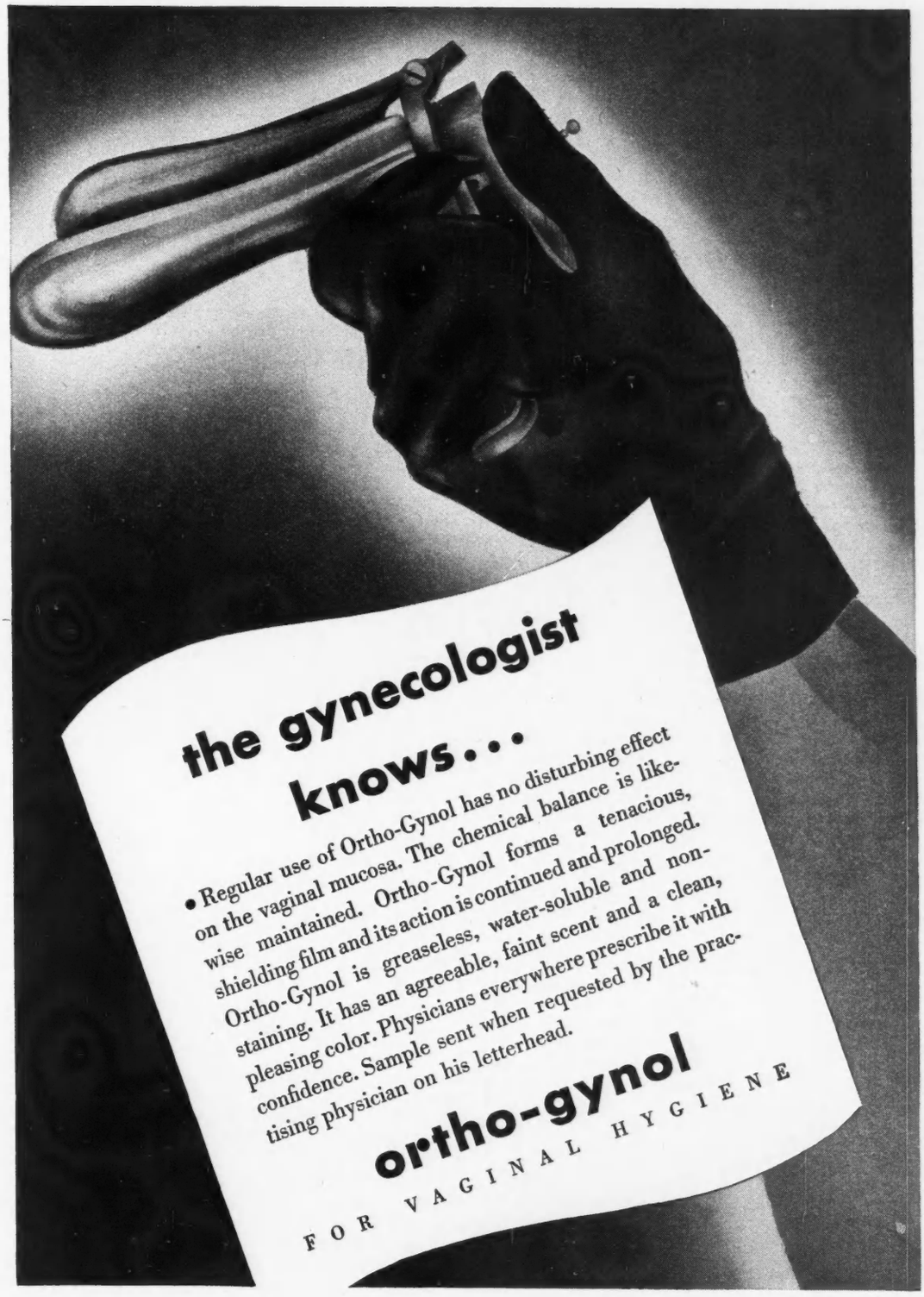
The description of the simple obstetrical operations is very concise and valuable, and the teaching throughout is of such a nature that the general practitioner, following instructions, would indeed be a safe obstetrician. The insistence that in the more dangerous conditions such as placenta prævia and accidental hæmorrhage the patient should be under the care of a skilled obstetrician and should be in hospital is highly commendable. In the description of placenta prævia mention might have been made of the fact that simple rupture of the membranes will suffice in properly selected cases.

This is a textbook which can be highly recommended to students, and is not without value to the graduate.

A Textbook of Obstetrics. Frederick C. Irving, A.B., M.D., F.A.C.S., and William Lambert Richardson, Professor of Obstetrics, Harvard University Medical School. 533 pages, illustrated. Price $6.00. Macmillan Co., New York and Toronto, 1936.

The text is based on lectures covering normal and abnormal obstetrics delivered to students in the Harvard Medical School, and is a faithful presentation of the obstetrical teaching in that university and of the methods of practice in the Boston Lying-In Hospital. Beyond that, the book records the personal views of a brilliant teacher and successful obstetrician.

The first part, comprising 12 chapters, deals with normal obstetrics. The chapter on the physiology of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conception, is concerned with ovulation, menstruation, maturation and fertilization of the ovum, sterility, the formation of the decidua and implantation is entirely up to date, and covers a field too often neglected. The section on Diseases of the Newborn is written by Dr. Stewart H. Clifford, and that on Heart Disease in Pregnancy by Dr. Burton E. Hamilton.

The second part deals with abnormal obstetrics, including the pathology of pregnancy, labour, and the puerperium, and obstetrical surgery. To the student approaching the subject for the first time this book may be recommended as a lucid and concise exposition of present day obstetrics, and to the practitioner, as a statement of tried and tested methods to ensure safe and sane obstetrics. Dr. Irving is a sure guide along a path beset with pitfalls.

Abortion, Spontaneous and Induced, Medical and Social Aspects. F. J. Taussig, M.D., F.A.C.S., Professor of Clinical Obstetrics and Clinical Gynæcology, Washington University School of Medicine, St. Louis. 536 pages, illustrated. Price $8.50. C. V. Mosby, St. Louis; McAinsh & Co., Toronto, 1936.

In this monograph Taussig has dealt with abortion from every angle in a most detailed manner. The material assembled shows exhaustive research work with a compilation of opinions from many European and American authorities which he has adjudged, added to, and summarized so that the reader does not become confused in the wealth of detail. The book should be an excellent manual for the general practitioner, giving as it does the diagnosis, treatment, and underlying pathology in all types of abortion. It is well illustrated. The legal, ethical and theological aspects of the problem of abortion are fully discussed from an open-minded viewpoint; there is also a chapter on the abortaria of Soviet Russia as seen by the author in 1930, with added comment of the more recent developments of legalized abortion in that country with its unexpected sequelæ. Included in the volume is a chapter on abortion in animals. The laws covering abortion in most countries and all the United States are briefly summarized and numerous statistical reports are given.

Diseases of the Nervous System. S. E. Jelliffe, M.D., Ph.D., and W. A. White, M.D. Sixth edition, 1175 pages, illustrated. Price $9.50. Lea & Febiger, Philadephia, 1935.

The author has followed the same system of classifications as in the previous edition, published in 1930. The contents are divided into three parts: Part I—The Physico-chemical Systems. Part II—The Sensorimotor Systems. Part III—The Physical or Symbolic Systems. There are twenty-four chapters with many subdivisions, which make the work an excellent one for easy reference. There is also a complete index. Parts I and II are therefore devoted entirely to diseases of the Nervous System, while Part III is on Psychiatry. It is apparent that this very complete work has been carefully revised, and some portions rewritten. There are 497 illustrations and 12 plates. This book is well recommended for students and practitioners.

The Foot. Norman C. Lake, M.D., M.S., D.Sc., F.R.C.S., Senior Surgeon, Charing Cross Hospital. 330 pages, illustrated. Price $3.75. Baillière, Tindall & Cox, London; Macmillan Co., Toronto, 1935.

This is a well written volume in which the evolution and development of the foot are gone into in an interesting way. Wood Jones is quoted to the effect that mankind had no direct four-footed ancestry but that the stock from which he diverged was from the first arboreal. The chapter on the anatomy and physiology of the foot is very complete. There is a helpful chapter on footwear —ancient and modern—and also on gait and its abnormalities. There is a good article on abnormalities of the foot and an illuminating diagram showing accessory ossification centres. Foot-injuries, including wounds, sprains, dislocations and fractures, are treated adequately. Infections of the foot are given a separate chapter. The author takes issue with Sir Arthur Keith who has laid down that "nature never uses ligaments as prime supporters in the structure of the animal body; always muscles are used for this purpose. Ligaments serve only as safeguards; they come into action only when the muscular defense has broken down". The writer points out that the middle and external cuneiform bones could hardly be maintained in position by the postural tonus of any normal anatomical muscle. Good descriptions are given of bursitis and the various affections of the nails, corns, etc. The chapter on vasospastic lesions and their treatment when necessary by lumbar sympathectomy is very complete. The book on the whole is thorough and the treatment sound.

Principles and Practice of X-ray Therapy. F. Roberts, M.A., M.D., M.R.C.P., D.M.R.E., Physician in Charge of Douty X-Ray Clinic, Addenbrooke's Hospital, Cambridge. 214 pages, illustrated. Price 10/6 net. H. K. Lewis, London, 1935.

An excellent textbook for the use of the student and a source of stimulation and authoritative reference to the practitioner of radiotherapy. Dr. Roberts has packed an immense amount of information into a small volume. It is sufficiently detailed with regard to the physics and theory of x-radiation, yet the author never loses sight of the practical side of his subject, and physics, theory and practice are combined in such admirable proportions that one realizes both pleasure and profit out of the reading of it. Space given to the description of apparatus is deservedly limited, and when the author delves into the history of facts and theories of radiation and biological effects he always does so with an end in view, and he describes the old in a few graphic sentences that illuminate and clarify the new.

The modern conception of the measurement of quantity and quality of x-rays is well explained and surface and depth dosage is fully discussed. The author, who is a strong advocate of the use of several small converging beams in the treatment of deep seated lesions, describes a unique method for the localization and direction of the x-ray beams, by the use of the "flexible curve", an instrument used by engineers and surveyors. The time factor and the expression of dosage are well handled, and the chapters on the action of x-rays on normal tissues and the general principles in the treatment of malignant disease are very instructive.

The concluding chapters of the book deal with the author's method of treating malignant and benign diseases. Some of the methods used are new in the experience of the reviewer, but one feels that the author has stated his case well and that these methods are worthy.

This book is a valuable addition to the literature of radiology.

Index of Treatment. Eleventh edition, edited by R. Hutchison, M.D., LL.D., F.R.C.P., Consulting Physician, London Hospital. 1020 pages, illustrated. Price $12.00. John Wright & Sons, Bristol; Macmillan Co., Toronto, 1936.

We have called attention to previous editions of this book and can add but little now except a repetition of our commendation. The latest advances in therapeutics have all been incorporated, with a good deal of rewriting of some sections. In spite of these additions and changes the book has been kept within reasonable limits of size, and has actually been shortened by a few pages.